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THE SWORD OF DAMOCLES*

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Homer relates to us the story of Damocles, a courtier of ancient Greece, whose king Dionysius demonstrated to him the dangers of a ruler's life by seating him at a banquet with a sword hanging by a hair over his head. The doctor of today is a modern Damocles, threatened by the sword of medicolegal action being dropped upon him by a disgruntled patient. Malpractice suits constitute an occupational hazard of some magnitude, and the physician's professional and economic security literally hangs by a hair. No longer is he able to face each patient with the attitude of: "How may I help my brother?" but rather with the reservation of: "How can I keep him from suing me?"

Claims for damage from malpractice may be said to have originated in the code of Hammurabi in 2250 B.C., which prescribed a fine against the physician of 100 solidi, or $225.00, in the event of an injury to a nobleman during the performance of a venesection. The physician was not only deprived of his fee in the event the patient died, but he was turned over to the relatives of the deceased to do with as they saw fit. The loss of an eye from surgery resulted in amputation of the surgeon's hand. Obviously, in those days the surgeon had to be endowed with a high degree of personal courage as well as professional skill. This stringent attitude was relaxed somewhat over the centuries, but today the pendulum has started to swing back.

The first reported case of medical professional liability was filed in Connecticut in 1794. Up to 1900, only 224 such suits were known in the entire country, and 30 years ago they were still a rarity. Today it has been estimated that from six to nine thousand suits are filed annually against physicians and hospitals. The number of suits seems to vary geographically from one out of eight doctors in the country at large to one out of five in New York and the District of Columbia. The peak is reached in California, where one out of four has been sued. Curiously enough, this seems to parallel the fees charged in the corresponding areas. In all, it is estimated that 18,500 living members of the American Medical Association have been sued for malpractice during their professional lives.

Not only has the incidence of malpractice suits increased, but the size of individual judgments has skyrocketed to an average of $75,000.00 in this era of the "more adequate award." The prevailing philosophy seems to be: although money may be a poor substitute for the catastrophe of injury, a large sum and only a large sum can fully compensate the injured. Because of the exorbitant sums awarded by juries today, the $25,000.00 medical protective insurance, formerly considered adequate, has now had to be increased to $250,000.00 or even higher. The cost of this insurance has risen steadily from $35.00 in 1928, my first year in practice, to $164.70 this past year. Malpractice action against members of the medical profession has become "Big Business," costing physicians one million dollars annually, with the ultimate cost to physicians and patients reaching fifty million dollars.[6] Whereas the plaintiff is awarded a verdict in only one out of eight cases coming to trial, the damage to the defendant doctor by

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the unsavory publicity is inestimable. Moreover, Morris estimates that of all malpractice suits, 95 per cent are competely unjustified, 3 per cent justified, and the remaining 2 per cent pure shakedowns.

What are some of the factors that have brought about this situation? First of all, medicine, unlike chemistry, is not an exact science. The mixing of two compounds in a test tube under controlled conditions always results in the same predictable reaction. Patients, on the other hand, vary in their individual reactions to drugs and treatment in a variety of ways that cannot be anticipated.

The doctor himself is not the infallible little god depicted by Hollywood, TV, and many lay magazines, and which sometimes he himself is convinced that he is. After all, he is only a human being who may be faced frequently with grave emergencies requiring split-second decisions. These decisions may have to be rendered when he is physically exhausted, or in the early morning hours when no man is at his best. The sooner this illusion of his infallibility is dispelled in the medical as well as in the lay mind, the fewer will be the number of suits filed.

As the practice of medicine has become increasingly complicated, an ever increasing number of assistants are needed. For instance, it has been estimated that intracardiac surgery may involve as many as 75 persons in diagnosis and treatment. The courts have held that the physician is responsible for the acts of all ancillary personnel, including hospital employees, even though he may have little voice in their selection or training. Instances such as an untrained nurses aid burning an unconscious patient with a hot water bottle have resulted in damages against the doctor. Suits have been brought against doctors 3000 miles away from the scene of the supposed act of negligence. Others have been sued for referring a patient to a specialist who was allegedly negligent in his handling of the case.

A decisive factor is the suit-mindedness of the public, possibly brought about by the increasing frequency of automobile accidents and the attendant liability. Studies have shown that the majority of malpractice plaintiffs have previously been involved in other civil actions. The idea has been developed in the minds of many people that someone is always responsible when something goes wrong and can be made financially responsible for the damage, real or fancied, which may result. Part of this may spring from the philosophy of the communal responsibility for the individual, from the cradle to the grave, introduced by the New Deal.

The plaintiff's chance of winning his case has been greatly strengthened in some areas by the court's over-extension of a principle legally known as *res ipsa loquitur* or "the thing speaks for itself," and against which the physician is completely defenseless. Practically it has been distorted into a rule of sympathy. The legal profession piously maintains that this principle is its only means to combat the "conspiracy of silence" that exists in the medical profession when a brother practitioner is sued. Ordinarily, expert opinion is required to support the plaintiff's charge of negligence in a malpractice suit. The present difficulty of obtaining this is almost equivalent to a defendant being unable to obtain legal counsel for his defense in a criminal action—a right guaranteed to him by the constitution. The use of the principle of *res ipsa loquitur* allows any jury to decide a medical question without the benefit of medical testimony. Conclusions reached by this principle are often more fallacious than convictions for criminal offenses based upon circumstantial evidence. A good illustration is the case cited by the Drs. Wilson in which a teen-age male with an acute appendix was advised to have a spinal anesthetic for the appendectomy because of a concurrent respiratory infection. This was refused and a general anesthetic given. Seven days later an ascending myelitis beginning in the lumbar area developed, with almost total paralysis of the lower extremities caused by the Guillain-Barré syndrome. If a spinal anesthetic had been used and a suit brought against the doctor on the basis of *res ipsa loquitur*, would not a jury, composed even of surgeons, have said that this unfortunate complication was the result of infection introduced at the time of the spinal puncture? Review of the literature reveals that even epidural abscesses can occur as a complication of acute appendicitis due to lymphatic spread. The end result of the application of this "rule of sympathy" would be a modern Hammurabian code requiring the surgeon to be a guarantor of a good result in every operation. This is obviously impossible.

The increasing number and complexity of

TABLE 1

Primary reasons for claims

Reasons	Percentage
Poor operative reasons	24.7
Poor medical results	19.3
Error in diagnosis	10.1
Foreign bodies left in patient	8.6
Burns	7.9
Assault, lack of consent, abandonment	6.8
Miscellaneous	22.6

TABLE 2

Secondary reasons for claims

Reasons	Percentage
Careless comment by another physician	25.2
Action prompted by an attorney	15.4
Claims brought for financial profit	13.8
Patient had mental disability	7.9
Action prompted by relatives	7.2
Patient was alcohol or narcotic addict	2.9
Patient had another disease or disability which progressed badly	2.1
Miscellaneous	25.5

modern drugs add still another factor, that of unsuspected idiosyncrasy to a particular drug. Many suits have been instituted against doctors because of unexpected drug reactions. For instance, in the District of Columbia an unemployed woman brought suit against a doctor for two million dollars because of a penicillin reaction occurring during routine treatment in a case of gonorrhea. Because of the common use of antibiotics in the animal and poultry industries, people can now be sensitized to these substances through foodstuffs. A careful and detailed history of drug sensitivity should be an integral part of every hospital record, and a list of such allergies posted conspicuously on the chart. Woe to the physician who overlooks this note!

Specifically there are a number of reasons for malpractice claims. These are listed in tables 1 and 2, which are self-explanatory.

What effect does this rising tide of legal action have upon medical practice? Certainly the stand of the courts at present can stymie advances in medical knowledge and the development of new techniques and new treatments. It would require a very bold individual to try a new type of treatment in his community knowing that a poor result, whether real or fancied, might cost him his life's savings. In a speech before a group of 200 research administrators, attorney Irving Ladmir pointed out that legitimate research as well as quackery, justifiable nonconformance and negligent deviation may hang upon the gibbet of malpractice. The recent example of a prominent west coast vascular surgeon being sued for a large sum because of a complication from an aortography done by his chief resident would tend to discourage others from attempting to train interns and residents. The trial court held the surgeon himself responsible for the result. This precedent could be interpreted to mean that the surgeon himself must personally perform every trivial act associated with the care of a patient from the initial enema, to include all blood counts and laboratory tests, down to the final surgical dressing at the time of discharge. This would decrease the patient load of the individual doctor in the face of an already critical shortage of qualified surgeons. The steadily mounting cost of medical care is further increased by unnecessary x-rays and laboratory procedures and superfluous consultations, all done to avoid charges of negligence rather than to increase the quality of medical care. A good example of this is the present hospital regulation requiring examinations by a pathologist of such surgical specimens as hernial sacs to prove "that the surgeon actually treated the hernia." Of course, this extra cost is added to the patient's bill!

The threat of malpractice action prevents many physicians from stopping at the scene of an accident to render the first aid which might well be life saving. Courts have held that rendering such first aid represents a contract between the victim and the doctor, who can then be held financially responsible for any alleged poor result. California has recently enacted legislation which would curb this injustice.

In addition to malpractice, a disgruntled patient can attack a physician's financial security on other grounds. Lack of so-called "proper consent" can be made the basis of a suit for assault, as occurred recently in a case at Morrilton, Arkansas. The surgeon who was called upon to treat a hunter for a severe hemorrhage from a gunshot wound of the thigh found a false aneurysm due to an injury of the femoral artery. In

order to control hemorrhage it was necessary to ligate the artery, with resultant gangrene of the leg. The physician was sued for assault on the grounds that he had contracted to remove the bullet but did not obtain specific consent to ligate the artery. No question of malpractice was involved, since the patient conceded that the treatment had been correct. An $80,000.00 judgment, however, was rendered by the jury for the plaintiff. This was not covered by the doctor's malpractice insurance since the award was for assault, a criminal offense. Had the patient been allowed to bleed to death, the surgeon might have been sued for malpractice, but then at least the insurance company would have borne the financial loss. In addition, this loss is not income tax deductible because of the criminal nature of the offense. Fortunately, the Supreme Court reversed the decision and the case was settled out of court for a lesser sum. A Tulsa, Oklahoma, anesthesiologist was penalized $125,000.00 for assault for not obtaining proper consent for spinal anesthesia, the consent having been obtained after the preoperative medication had been given. The court ruled that proper consent had not been obtained because the patient was under the influence of drugs at the time. This unfortunate physician is financially ruined for the rest of his life, because it is impossible to save such a sum as this without the benefit of income tax deduction.

Lack of proper consent has also been held to occur when the surgeon fails to warn the patient before surgery of all possible complications that might follow the operation. Elaborate consent forms, such as the one recently advocated by Rieser,[5] have been devised to cover every contingency. To discuss all this with the patient would be tantamount to reading a medical encyclopedia to him. This would not only be time consuming, but it would cause any sensible patient to lose confidence in his doctor and to refuse to sign the consent form. On the other hand, the usual blanket type of consent which allows the surgeon to use his best judgment, depending upon conditions encountered at the time of surgery, has been held not binding upon the patient. This leaves the surgeon exposed to legal action for assault.

This ground is particularly treacherous when the patient is a minor. A case occurred a few years ago in Oklahoma City in which a teen-age boy was injured in a traffic accident, with a resultant severely mangled leg and hemorrhage. The parents could not be reached, so the surgeon asked for and received consultation from two eminent surgeons, who agreed that immediate amputation was the only way of saving the patient's life. This was done, but the surgeon was later successfully sued by the parents for assault. Another dangerous situation is that in which a child is living with one parent in a broken home, or possibly in that of a close relative, or even a friend, and the question of emergency surgery arises. Despite the fact that the child's life may have been saved by the treatment, courts may rule in favor of an estranged parent who sees an opportunity for making a fast buck. Pathologists particularly must be on guard to see that consent forms are properly signed before performing necropsies. In this day of substitution of parts of the body, the surgeon may be held liable for assault by the donor or his next of kin. We have been threatened with a suit by a donor of skin, an aunt of a severely burned child, who alleged that her donor site has since caused her "extreme pain and suffering."

Action against a physician for libel, based upon a report made to an insurance company in which the defendant doctor suggested that the patient was a malingerer, has resulted in damages for the plaintiff. Probably with the present trend of the courts, the innocuous phrase "no objective evidence of injury" could be interpreted likewise. Adverse decisions have been rendered against physicians who apparently were testifying on court's orders. In the case of Alvin *versus* Morgan, a physician was penalized for telling an unwed girl's mother that she was pregnant.[4]

Most malpractice insurance policies carry a clause exempting them from damages arising from malpractice because of treatment administered while a doctor was under the influence of drugs or alcohol. What drug and how much alcohol would allow the insurance carrier to escape his financial responsibility? Suppose, for instance, that a surgeon has been called to the emergency room to suture a laceration and a poor result ensues. Is the antispasmodic he may have taken for his ulcer a narcotic because it contains a small quantity of phenobarbital? How many of us are called occasionally for just

such a case after taking a preprandial highball? Are we then under the influence of alcohol?

Operations for sterilization offer another fertile field for the enrichment of conniving patients and lawyers. No consent form has yet been brought out that will adequately protect the hospital or doctor in cases in which sterilization has been done for any reason other than the health of the individual. Very few physicians today are willing to jeopardize their financial and mental security by performing sterilization operations for any other reason, no matter how justified they may be.

The term "hospital infection" has become popularized by many lay magazines as referring to an infection contracted within the hospital walls, and to the uninitiated, meaning negligence on someone's part. Another bonanza may thus have been created for those who have no scruples concerning how they may acquire a dollar, because the very term puts two strikes against the doctor or hospital before the case is filed, if the principle of *res ipsa loquitur* is employed. This threat has resulted in greatly increasing the *per diem* cost of hospital care, because of the necessity on the part of the hospital for taking preventive measures considered necessary to circumvent a charge of negligence. This involves such things as phage typing of all personnel, specially filtered air conditioning systems for operating rooms, ultraviolet radiation, etc., provisions which are extremely expensive. Nonprofit hospitals were formerly immune against civil actions unless they carried insurance for such protection, in which case the insurance carrier could be sued. Since the cost of such insurance is prohibitive, the answer is to carry no insurance, in which case the physician then becomes liable.

This is not the time or the place to discuss hospital infections, but we would like to call your attention to an article by Barnes appearing in the July issue of *Surgery*, in which the incidence of infection in The Massachusetts General Hospital following two standard operations, herniorrhaphy and hysterectomy, is compared yearly over a period of 21 years. During this period of time there was little variation in the rate, namely, 5.2 per cent for hernias and 3.8 per cent for hysterectomies, showing that the problem of infection is not a new one. When one reflects that if he does 100 hernia operations, for which he might expect to receive $15,000.00, and balances this against the 5 infections that he may have, for which juries may award $75,000.00 each, it is easy to see that surgery is an extremely poor speculative risk.

I have not listed all the legal dangers to which the surgeon is exposed, but have cited a few examples to illustrate the point that I am trying to make; namely, that we must often compromise good medical and surgical judgment in order to avoid costly legal entanglements. Watts, in an informal questionnaire among his conferees, found a decline in the use of spinal anesthesia; that radium therapy had been replaced by surgery for all forms of uterine bleeding; and that a general surgeon had discarded the radical Wertheim operation for cervical cancer and substituted simple total hysterectomy, in spite of the higher cure rate with the former, because of its high rate of ureteral fistulae.[9] Certainly this does not make for good medical practice!

How then can we avoid these costly and embarrassing situations? Several remedies have been proposed, none of which is completely trustworthy. First of all, we should try to maintain good doctor-patient relationships at all times and under all conditions, trying though they may be, because the spark that often sets off a suit is the desire for revenge on the part of the patient against his doctor for some real or fancied slight or insult. An aggrieved patient who may be worried about finances and not thinking clearly because of his illness is a dangerous person. Even extra attention on the part of the doctor may not always be sufficient. A markedly senile druggist, who was an arthritic taking large doses of cortisone, had a nephrectomy done for a large, almost inoperable carcinoma of the kidney. After removal of the sutures the wound broke open. Suit was brought against the hospital, which at that time was carrying insurance. A federal judge awarded the patient $4,000.00, the maximum allowed by the policy, in spite of medical testimony on the adverse effects of debilitation and cortisone upon wound healing. This suit occurred even with the attention the patient received at the hands of his physician during his entire convalescence, and a courtesy reduction in his fee because of his being a druggist. If a precedent like this becomes established, the ordinary rate of wound de-

hiscence that plagues each of us would again render surgery an exceedingly unprofitable field.

It has been recommended that in the event that a mistake is made in the treatment of a case, the situation should be frankly discussed with the patient or members of his family. This measure may not be effective, as in the case of one of my colleagues, in which the patient was wrongly positioned by the anesthetist before the arrival of the surgeon, with the result that a skin incision was made over the uninjured side. The mistake was discovered after the skin had been incised. The wound was sutured, the patient repositioned and the fractured hip nailed. The situation was explained to the patient and the family who, at the time, seemed reasonable. Convalescence was uneventful and an excellent end result obtained. This, however, did not prevent the patient from joining forces with a lawyer, who brought suit for negligence. The suit was settled out of court for $5,000.00, and the patient later told her family physician: "I sure hated to do that, but we have a nice nest egg in the bank now."

One of our best defenses is the keeping of adequate records on every case treated, no matter how minor. If there were only one good thing accomplished by the Joint Committee on Accreditation, it would be the insistence on complete records being made on every patient admitted. As an example, recently a member of my staff was threatened with suit by a patient who, after removal of an axillary gland, claimed that she had suffered extreme pain in her left shoulder "ever since the operation was performed." A review of the record on this patient showed that among her complaints listed before operation was "extreme pain in the left shoulder of several months' standing."

Of some value is the establishment of a good liaison with the legal profession so that its members may have some insight into our problems, and in return we may learn in what ways we irritate them. This makes for mutual benefit. Many county medical societies are now having annual joint meetings with local bar associations. It has been reported that even the noted malpractice specialist M. M. Belli has somewhat softened his attitude toward the medical profession after a recent stay in a hospital where he obtained first-hand knowledge of the obstacles that doctors have to face with every patient. He has made the suggestion that the hospitals themselves be self-insurers against injuries suffered within their walls by creating a fund for such exigencies, the cost of which would be met by a small extra *per diem* charge against each patient.[8] An alternative would be the sale of insurance policies to patients in the same manner as is being done with airline trip insurance. Unfortunately, human nature being what it is, frequent "shakedowns" by unscrupulous patients for fancied injuries would keep the fund exhausted. On the other hand, when there has been a definite injury to a patient because of real negligence, some form of reasonable compensation should accrue to the patient.

A member of our association, Dr. Ian M. Chesser of Tucson, described in a paper before this Congress in 1959 the review panel system set up by the Pima County Medical Society which, if widely adopted, would curb to a great extent this constant danger to which we are exposed. Eight members of the county medical society and a similar number of members of the local bar association, together with the patient and his attorneys, and the doctor involved with his counsel, meet in an informal session to discuss the alleged malpractice claim. Two questions are asked. First: "Is there substantial evidence of negligence?" Second: "Has harm been done to the patient by the action?" If the answer is yes on either count, the plaintiff's attorney is free to take his case to court and to use medical witnesses who have appeared before the panel in presenting the case to the jury. This has resulted in weeding out the 95 per cent of cases that have no merit. Settlement out of court has usually followed when negligence or harm has been established. When it has not been proved, the case has been dropped. Of 12 cases heard by the panel in 3 years, none has come to court. While attorneys are not bound by the panel's decision, they like the system because they are thus able to get expert testimony if the need arises and to avoid unnecessary work if the case has no merit.

At the present time 31 states have claim review programs on a state or county level, and 21 states have claim prevention programs according to which a board reviews the claim for the purpose of advising the defendant or insurance carrier. However, even this method has been attacked, as shown by the recent filing of a two million dollar suit at Drumright, Oklahoma

against the defendant doctors, their legal counsel, and members of the local society who sat on the claim board on the grounds of conspiracy to defraud the plaintiff. Fortunately for precedent, the trial judge threw the suit out of court.

A bit of house cleaning is also in order within our own ranks. Tissue committees have made an important step forward in calling attention to unnecessary and incomplete surgical procedures in hospitals. This covers a large segment, since 70 per cent of the suits filed are the result of treatment administered in a hospital. There is, however, no check upon nonsurgical treatment or emergency room care. We must develop a constructive attitude among ourselves, so that we will not feel the reluctance that we do at present in calling attention to the substandard practice of a colleague. This particular phase, however, applies at present to only 3 per cent of the cases which are justified. By further cooperation with the legal profession it may be possible to win its confidence to the point at which the 95 per cent unjustified suits are not filed.

In this connection it is interesting to note the following suggestions which have been advanced by the National Association of Compensation Claimants Attorneys:[3]

1. There should be no nuisance suits filed and no nuisance settlements made by doctors or their insurance companies.

2. There should be some system of enabling attorneys to screen their clients' stories adequately before a claim is made upon the defendant doctor.

3. If a case reaches trial stage, there should be a method of obtaining fair, impartial and honest expert testimony for consideration by the jury.

4. There should be no publicity of malpractice cases.

5. There should be a joint physician-lawyer speaker's committee to participate in educational programs for physicians and lawyers at national, state and local levels.

There is much food for thought for us in these suggestions!

To these suggestions we should like to add a sixth; namely, the abolishment of the contingency fee basis upon which these cases are taken. Our neighbor to the north, Canada, requires that all malpractice claims be tried in court, and contingency fees are held to be illegal. As a result, the cost of malpractice insurance in Canada is $20.00 a year for a surgeon.

Lastly, we should deglamorize our profession. As was stated previously, the doctor is set upon a pedestal by the public and regarded as one who never makes a mistake, despite the fact that he is only a human being subject to the same errors of judgment that dog anyone who carries great responsibilities. Unfortunately, the "hindsightometer" has not yet been perfected. Some credit should be given to the fact that we are doing the best we know how to do, in the light of experience and training and under difficult and trying circumstances, circumstances over which we often have no control. A poor result inflicts upon a conscientious physician as much psychic trauma as it does upon the patient. If he has done all that he knows to do, he should not be expected to pay otherwise for a poor outcome. And just as certainly, if negligence through laziness, carelessness, avarice, personal animosity or incompetence is established and the patient is harmed, no one should refuse to admit that the extra expense the patient may be put to, or loss in earning power that he may sustain should be adequately compensated. We should all consider seriously the means and measures for erecting an adequate barrier between this sword of Damocles and our heads.

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SKIN LEVEL COLOSTOMY: EXPERIENCE WITH 53 CASES*

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For the patient requiring rectal excision, the establishment of a permanent colonic stoma is a devastating experience characterized by a feeling of social disintegration and physical wreckage. To minimize the duration of this transitory period of mental depression and adjustment to the abnormally located anus, the surgeon must strive to establish a colonic stoma that is not esthetically repulsive in the immediate postoperative period. The skin level type of colostomy with primary mucocutaneous anastomosis has proved to be superior to other types of colostomies. This paper is concerned with our experience with 53 cases of skin level colostomies during the last 4 years on the Proctologic Service at Ochsner Foundation Hospital.

TABLE 1

Indications for colostomy in 53 patients

Indication	Number
Benign lesions	
Stenosis of previous colonic stomas	7
Paracolostomy hernia	2
Perforation of colon	1
Rectovaginal fistula	1
Prolapse of colonic stoma	1
Total	12
Malignant lesions	
Adenocarcinoma	33
Epidermoid carcinoma	7
Lymphosarcoma	1
Total	41

HISTORY

In May 1951, Crider and Neubeiser[3] described the method of primary mucocutaneous suture that they had used since 1947 on 12 patients with good results. The same year Campbell and Schaerrer[2] described their technique of skin level colostomy, which they called "colostomy

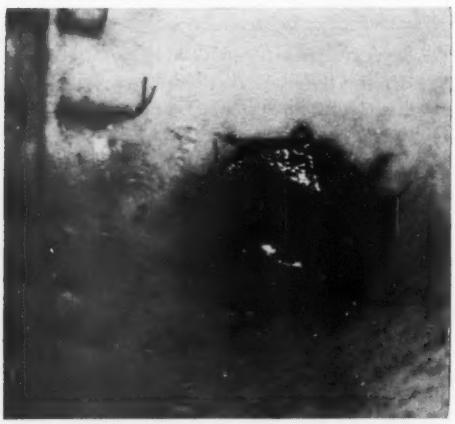

FIG. 1. Appearance of colonic stoma 5 days after skin level colostomy.

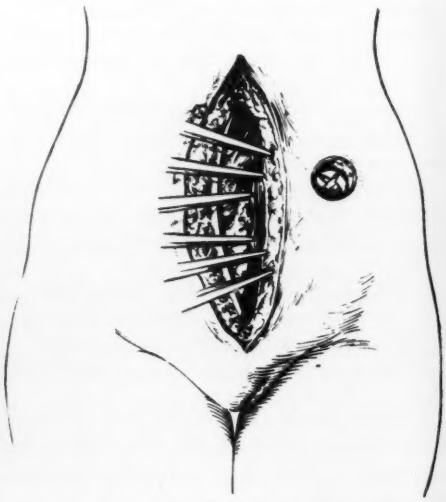

FIG. 2. Vertical perspective of the layers of the abdominal wall is maintained while the colostomy incision is extended through the abdominal wall by exerting traction on the peritoneum, anterior rectus sheath and subcutaneous tissues.

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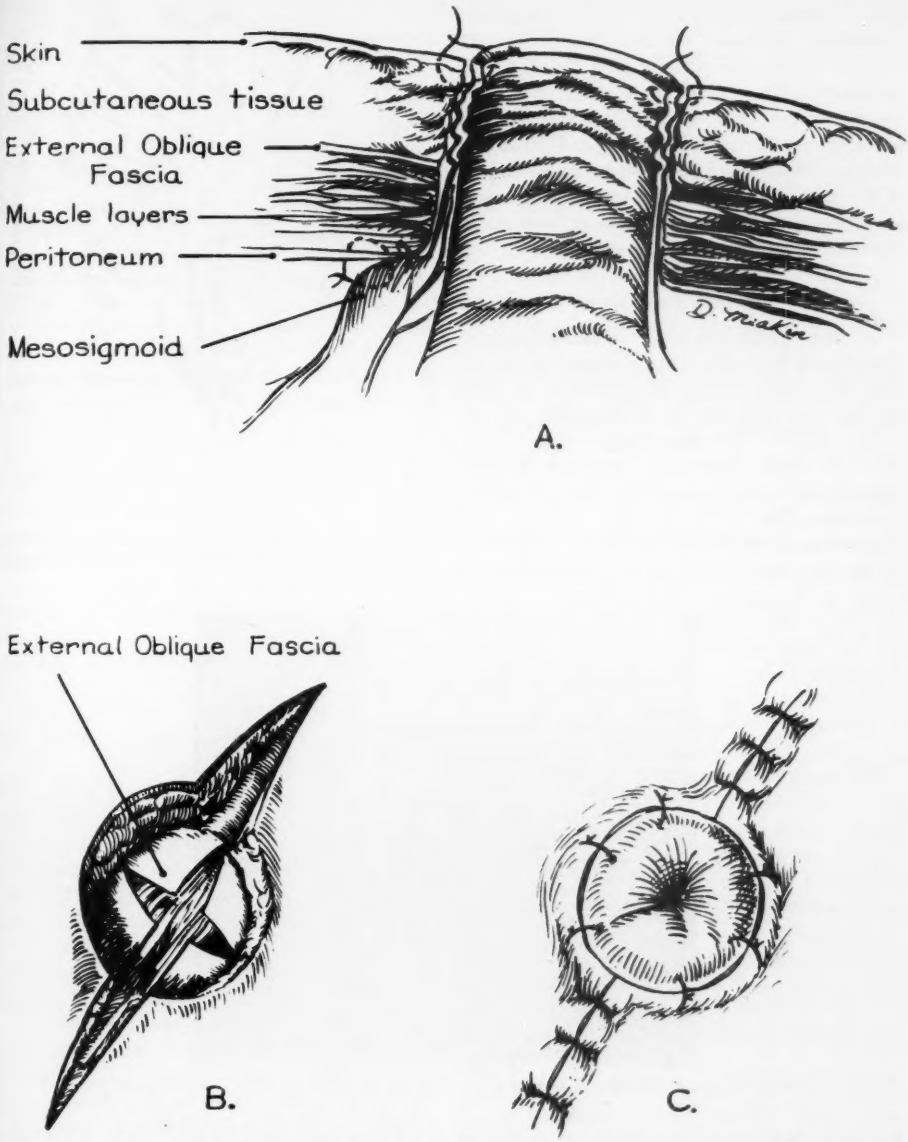

FIG. 3. *A*. The excessive length of intestine is tucked in the subcutaneous layer. Anastomosis by coaptation of the edge of the sigmoid is anchored to the peritoneum. *B*. Colostomy wound enlarged to permit pulling the intestine through the abdominal wall without injuring the blood supply. *C*. Closure of enlarged wound.

reconstruction," since it was done as a secondary procedure during the postoperative period. Since then Patey,[6] Butler,[1] Lemmer and Mehnert[5] and Donald[4] have elaborated on the surgical technique and have published uniformally good results. In July 1954, one of us (P. H. H.) performed a skin level sigmoid colostomy with primary mucocutaneous suture on a patient suffering

from a rectal stricture due to lymphopathia granuloma venereum on the Tulane Surgical Unit at Charity Hospital in New Orleans with excellent results (fig. 1). From that time until December 31, 1958 we have performed this procedure on 53 patients at Ochsner Foundation Hospital. Forty-one of these had malignant lesions of the rectum or anus, 5 had benign lesions and the remaining 7 required revision because of stenosis of previous colonic stomas (table 1).

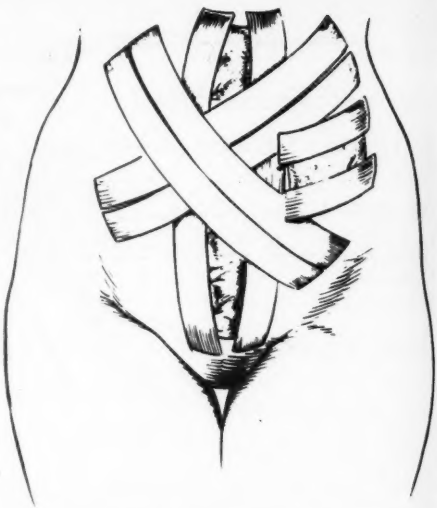

FIG. 4. Laparotomy wound and colostomy wound bandaged separately to permit inspection of colonic stoma.

SURGICAL TECHNIQUE

In the performance of abdominoperineal resection of the rectum, the abdomen is usually opened through a left paramedian or midline incision. The ideal site for a permanent sigmoid colonic stoma is through a separate incision in the left lower abdominal quadrant midway between the umbilicus and the anterior superior iliac spine. The colostomy site is selected and superficially outlined with a knife on the skin before the paramedian laparotomy incision is made. To construct the colonic stoma a circular incision is made, and a portion of skin measuring 1¼ in. in diameter together with the underlying subcutaneous tissue is excised. The vertical perspective of the layers of the abdominal wall is important for a well functioning colonic stoma and can be maintained by exerting traction on the peritoneum, anterior rectus sheath and subcutaneous tissue while the colostomy incision is extended through the abdominal wall (fig. 2).

FIG. 5. Appearance of a skin level colonic stoma several months after operation.

A cruciate incision is made through the external oblique fascia. The fibers of the internal oblique and transversalis muscles are separated by blunt dissection and the peritoneum is incised. The wound is sufficiently enlarged to receive the sigmoid colon. By insertion of an Ochsner forceps through the colostomy wound, the terminal sigmoid is clamped and pulled through the colostomy wound. Although we prefer to close the lateral gutter with interrupted sutures to

TABLE 2

Late complications in 53 colostomies

Complication	Onset	Revision of Colonic Stoma	Remarks
	year		
Stenosis	1	Yes	Irritable bowel
Stenosis	2	Yes	Irritable bowel
Paracolostomy hernia	1	No	Asymptomatic
Paracolostomy hernia	½	Yes with herniaplasty	Recurrent hernia
Paracolostomy hernia	1	No	Asymptomatic
Prolapse of colonic stoma	None		

prevent herniation of the small intestine, in some patients the space is large and this may not be necessary.

The mesentary of the sigmoid is anchored to the peritoneum of the anterior abdominal wall at the site of the colonic stoma (fig. 3*A*), and the appendices epiploicae may also be sutured, if present, but suturing of the sigmoid to the peritoneum about the circumference is not necessary. The intestine is brought out through the wound so that it projects 2 to 4 cm. above the skin level, depending upon the obesity of the person. After the paramedian incision has been sutured, the sigmoid is severed below the forceps and the blood supply is thoroughly checked at this time. The redundant intestine protruding above the skin is tucked between the skin and the fascia (fig. 3*A*). The edge of the intestine is sutured flush to the skin with interrupted, atraumatic, 3-0 chromic sutures on a cutting needle. The edges of the skin and of the intestine are coapted with a simple through-and-through suture that is loosely tied (fig. 3*A*). In the immediate postoperative period the colonic stoma is flush with the skin (fig. 1).

In obese patients with excessive fat in the mesosigmoid and large appendices epiploicae, the skin and external oblique fascial incisions are extended parallel to the fibers to permit pulling the intestine through the wound without injuring the blood supply (fig. 3*B*). The enlarged incision is closed with interrupted sutures so that the index finger can be inserted between the colonic stoma and the wound without a feeling of tightness (fig. 3*C*). The operator must be careful not to twist the intestine on its long axis.

The colonic stoma is covered with a Vaseline gauze dressing and a 4-in. by 4-in. sponge as a separate dressing from the paramedian wound (fig. 4). This separate dressing permits inspection of the colonic stoma for viability and retraction without disturbing the abdominal wound dressing.

ADVANTAGES

In the immediate postoperative period, the more esthetically satisfactory appearance of the colonic stoma considerably minimizes the degree of mental depression of patients and contributes a great deal toward early acceptance of the colonic stoma. Because the colon is patent, the gas and feces will be free to pass through the colonic stoma. This obviates the discomfort of intestinal distention and colonic cramps and permits early removal of the nasogastric decompression tube. Teaching the patient to accept the responsibilities and to establish the routine of emptying the colon by administration of small saline enemas can be started at an earlier date without fear of retraction of the colonic stoma. Most patients are hesitant and unwillingly accept management of a colonic stoma that protrudes above the skin level because of the severe edema and the seropurulent exudate over the surface of the exposed colon in the immediate postoperative period.

The greatest advantage of this technique is the primary healing of mucocutaneous anastomoses and absence of inflammatory scarring (fig. 5). With skin level colostomy, digital dilation of the stoma is unnecessary. In colonic stomas that protrude above skin level the huge protruding mass will undergo involution, and finally, after several weeks, the mucosa will reach skin level. In the process of maturation, serositis will result in intestinal edema and excessive circumferential scarring at skin level and in the sub-

mucosal layer of the intestine, resulting in stenosis and dysfunction in some colonic stomas.

RESULTS

There were no operative deaths among our 53 patients. During the immediate postoperative period such complications as retraction, necrosis of the terminal intestine, infection, dehiscence of the colostomy wound, bleeding or obstruction did not occur.

Five patients could not be traced for follow-up. The remaining 48 patients have been observed at intervals for periods ranging from 6 months to 4 years. The late complications are listed in table 2. Stenosis developed in 2 patients, 1 and 2 years, respectively, after the initial operation that required revision. These two patients had an "irritable bowel" syndrome and they continued to have frequent loose stools, making regulation of the colonic stoma impossible. In 3 patients a paracolostomy hernia developed, 1, 6 months postoperatively and 2, 1 year after operation. In only one patient was it necessary to perform a hernioplasty with revision of the colonic stoma at that time. The other 2 patients were asymptomatic and operation was not necessary.

SUMMARY

Skin level colostomy is a superior technique for constructing a colonic stoma. Because it is esthetically not repulsive to the patient, the duration of mental depression and adjustment that patients with colonic stomas must overcome during the immediate postoperative period is reduced.

This technique was employed in 53 patients with benign or malignant lesions of the rectum or colon. There were no immediate postoperative complications of retraction, bleeding or infection. Forty-eight of the 53 patients have been observed at intervals for periods ranging from 6 months to 4 years. Stenosis developed in 2 and paracolostomy hernias in 3. All other patients have well functioning colonic stomas.

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INFUNDIBULAR PULMONIC STENOSIS WITH INTACT VENTRICULAR SEPTUM*

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Despite an increasing number of reports to the contrary, infundibular pulmonic stenosis with intact ventricular septum is considered a rare congenital anomaly. In 1959, 3 patients with this condition were encountered in a series of 60 patients undergoing open correction of congenital cardiac lesions at the University of Oklahoma Medical Center. A discussion of the findings concerning and, the management, of these patients seems warranted in order to emphasize the special problems encountered in their diagnosis and treatment.

CASE REPORTS

Case 1

D. C. (U.H. No. 181-532), a 12-year-old white boy, was first noted to have a heart murmur in infancy. During early childhood, his development was somewhat slow, and the patient fainted after severe exercise on several occasions. At 8 years of age, cardiac catheterization revealed a right ventricular systolic pressure of 120 mm. Hg and a pulmonary artery systolic pressure of 15 mm. Hg. The transition in the pressure tracing between pulmonary artery and right ventricle was noted to occur rather low and, in retrospect, the pressure tracing suggests infundibular stenosis. Because no definite infundibulum was demonstrated, a diagnosis of valvular pulmonary stenosis was made. There was no evidence of shunting. Transventricular pulmonary valvulotomy was carried out several months later. At the time of operation, no change in the systolic thrill in the pulmonary outflow tract was noted. After the operation, the parents reported some improvement in exercise tolerance; however, the patient continued to have occasional loss of consciousness after severe exercise. Repeat catheterization was performed 6 months before the present admission.

Physical examination. At the time of admission examination revealed an underdeveloped, white boy. His blood pressure was 110/70. There was no cyanosis. There was slight prominence of the left chest with a left parasternal lift. A harsh systolic thrill was palpable along the left sternal border. There was a grade IV harsh systolic murmur which was maximal at the second and third interspaces along the left sternal border. The murmur radiated well to the neck, back and axilla. The second sound was diminished and split.

Laboratory findings. The hemogram and urinalysis were normal. Chest x-ray showed right ventricular enlargement. The pulmonary artery segment was decreased, and the pulmonary vascular shadows were normal. Electrocardiogram revealed right ventricular hypertrophy, and an incomplete right bundle branch block which had not changed after the previous operation. At cardiac catheterization, the right ventricular systolic pressure was 164 mm. Hg. A simultaneously recorded branchial artery pressure was 116/60 mm. Hg. Because of technical difficulties no attempt was made to enter the pulmonary artery. Brachial artery saturation was 95 per cent. Dye curves with right ventricular injection showed no evidence of right to left shunting. A diagnosis of pulmonary stenosis with intact ventricular septum was made.

A second operation was carried out on April 8, 1959, with the use of the midline sternum-splitting incision. The cavae were intubated through the right atrium and an arterial catheter was placed in the right superficial femoral artery. A modified DeWall-Lillehei bubble oxygenator with sigma-motor pump was used, and after establishment of satisfactory bypass, a normal pulmonary valve was exposed through an arteriotomy just above the valve ring. A finger could be passed through the valve and an infundibular stenosis palpated. Right ventriculotomy was carried out, and an infundibular muscular diaphragm excised with scissors. After completion of the cardiac repair and after discontinuing bypass, the right ventricular systolic pressure was 45 mm. Hg, and the brachial artery systolic pressure was 110 mm. Hg (fig. 1).

Postoperative course. The patient's postoperative course was uneventful. He was discharged from the hospital 3 weeks after operation in good

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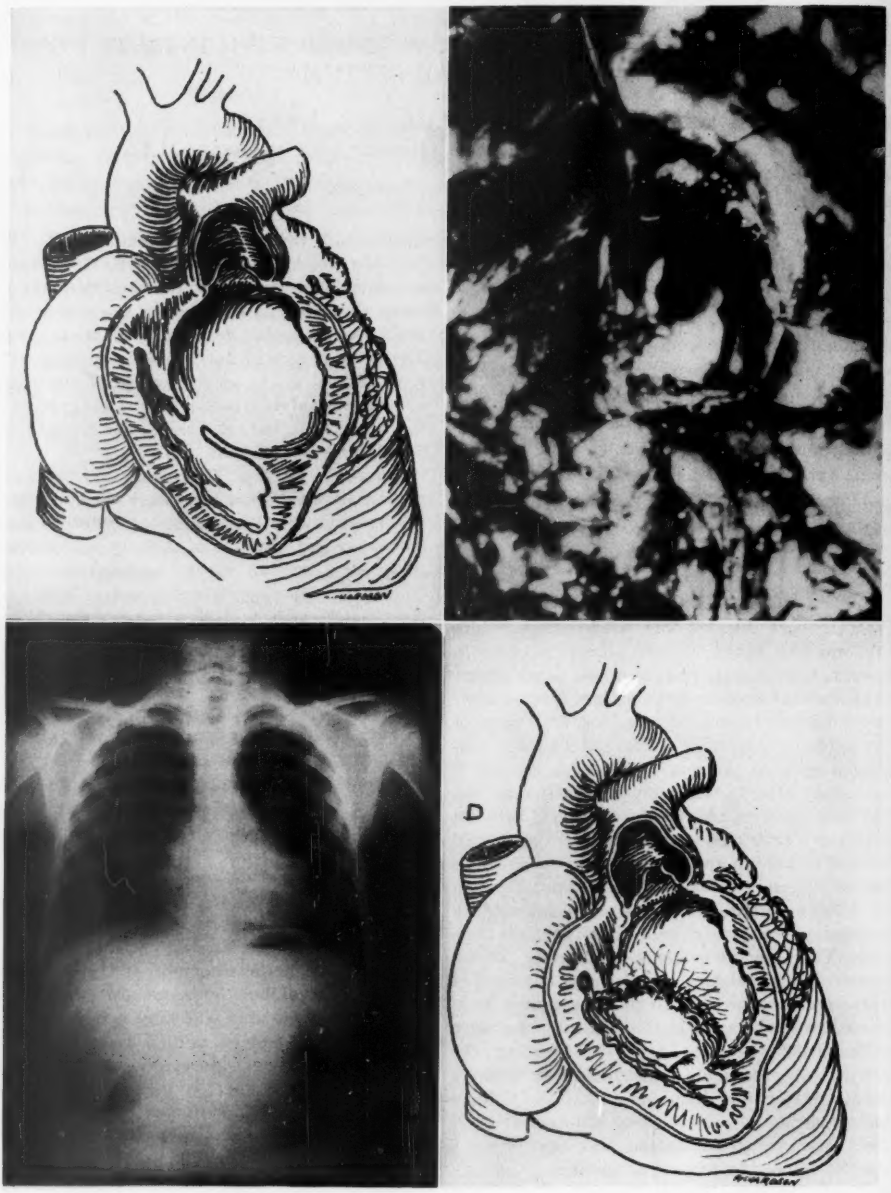

FIG. 1. Case 1. *Upper left*, drawing demonstrating site and nature of defect; *upper right*, operative photograph of right ventriculotomy. Small opening at *center* is actual opening in infundibular diaphragm. *Lower right* shows repair of defect.

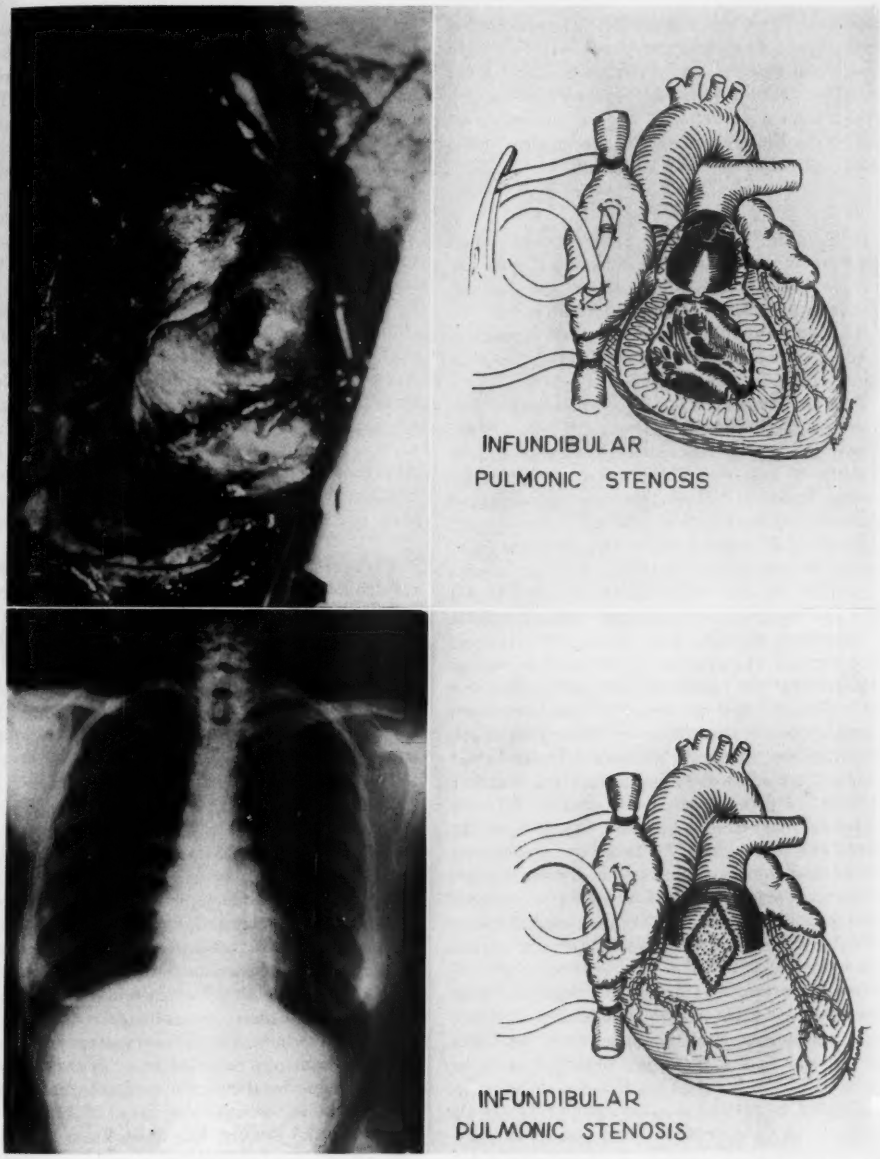

FIG. 2. Case 2. *Upper left*, photograph demonstrating external appearance of infundibular chamber. Drawing at *upper right* demonstrates site of pathology. *Lower right*, drawing illustrating placement of outflow patch.

condition. There was dimunition of the precordial thrill, but a loud murmur persisted. The right ventricular impulse was diminished. Chest x-ray revealed a normal heart size 3 months postoperatively. Repeat catheterization has not been carried out. Electrocardiogram shows less right ventricular hypertrophy.

Case 2

P. C. (U.H. No. 264-032), an 18-year-old white girl, was first found to have congenital heart disease during a febrile illness at 16 years of age. She gave a history of easy fatigability, but this had not prevented her from leading an active life. Cardiac catheterization was carried out 3 months before admission for surgery.

Physical examination. The patient was a well developed, slightly obese, white girl who did not appear acutely or chronically ill. There was no evidence of cyanosis. There was a left precardial cardiac impulse with no thoracic deformity. A grade V systolic murmur was audible to the left of the sternal border in the second and third interspaces, and there was a palpable thrill in the same area. The murmur was well transmitted to the neck and back. The second sound was not audible.

Laboratory findings. Hemogram and urinalysis were normal. The chest x-ray revealed no cardiac enlargement; the pulmonary arterial segment was not enlarged, and pulmonary vasculature was normal. The electrocardiogram showed right axis deviation and moderate right ventricular hypertrophy. At cardiac catheterization right ventricular systolic pressure was measured at 127 mm. Hg· pulmonary artery pressure was 15 mm. Hg. There was no evidence of a shunt by oxygen saturation or dye curves. No technically satisfactory pulmonary artery to right ventricle withdrawal tracing was obtained. A diagnosis of pulmonary valvular stenosis with intact ventricular septum was made.

Operation was performed on June 17, 1959. The heart was exposed through a midline sternum-splitting incision and the presence of an infundibular chamber was readily observed upon inspection of the heart. This thin walled chamber was noted to fill and contract slightly after the body of the right ventricle, and there was a constricted area approximately 5 cm. below the pulmonary valve ring. A harsh thrill was present in this portion of the ventricle and did not change in character distal to the pulmonary valve. The cavae were cannulated through the right atrium and arterial cannulation of the right femoral artery was carried out. Connections were completed to the modified DeWall-Lillehei bubble oxygenator with a sigmamotor pump, and after establishment of satisfactory cardiopulmonary bypass, the infundibular chamber was opened, exposing a thick muscular diaphragm separating the infundibulum from the right ventricle. The opening in this diaphragm was estimated at 8 mm. in diameter. After a large amount of the infundibular muscular ridge was trimmed away with forceps and scissors, there still appeared to be narrowing of the right ventricular outflow tract, and accordingly, a diamond shaped patch of laminated Ivalon and Teflon was sutured into the defect. Aortic cross clamping was carried out for 17 minutes, and after release of the aortic cross clamp ventricular fibrillation occurred. Sinus rhythm was readily restored with electric shock. Good cardiac action was maintained, and after removal of the cannulae, the right ventricular systolic pressure was 25 mm. Hg, and the brachial artery systolic pressure was 100 mm. Hg. No flow measurement was carried out at this time. The bypass was carried out for a total of 27 minutes and perfusion was considered satisfactory (fig. 2).

Postoperative course. The patient's postoperative course was uneventful. The thrill and murmur were lessened in intensity and there was no change in heart size on x-ray. After discharge from the hospital, the patient continued to improve and was asymptomatic at the time of this report. Postoperative catheterization has not been carried out. The electrocardiogram no longer shows right ventricular hypertrophy and the axis is normal (fig. 3).

Case 3

K. M. (U.H. No. 281-427), a 22-year-old white man, was noted to be slightly cyanotic shortly after birth. Early development was retarded, and the patient had numerous respiratory infections in infancy and childhood. He was first seen at the Medical Center 4 months before the present admission, at which time he was found to have congenital heart disease, mental deficiency, cryptorchidism and nontoxic diffuse goiter. Extensive endocrine work-up revealed no evidence of recognizable endocrine deficiency syndrome, and during this admission, cardiac catheterization was carried out. The patient had never been employed and had led a very sedentary existence.

Physical examination. The patient was a poorly developed, thin white man who appeared younger than 22 years of age. His blood pressure was 130/60. On examination of the head and neck, a high arched palate was noted. There was an increased anteroposterior diameter of the chest with prominence of the left precardium. There was a left parasternal tap. A systolic thrill was

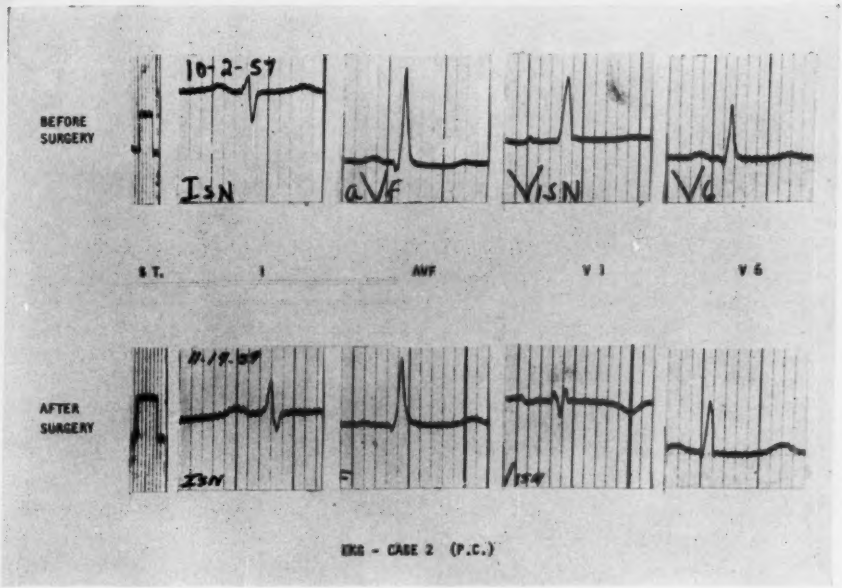

FIG. 3. Case 2. Electrocardiogram before and after surgery, showing virtual disappearance of right ventricular hypertrophy.

palpable to the left of the sternum in the second and third interspaces, and a grade IV harsh systolic murmur was audible to the left sternum. This murmur was well transmitted to the axilla and neck. The second sound was diminished in the pulmonic area. There was femine hair distribution with cryptorchidism. Examination of the extremities revealed enlargement and hyperextensibility of both knees and elbows.

Laboratory findings. Routine hemogram and urinalysis gave normal findings. Chest x-ray showed enlargement of the right ventricle. The pulmonary artery was prominent, and the vascular shadows were considered to be within normal limits. Electrocardiogram demonstrated first degree atrioventricular block, marked right axis deviation and marked right ventricular hypertrophy. Catheterization, on April 16, 1959, revealed a right ventricular systolic pressure of 165 mm. Hg, pulmonary artery systolic pressure of 20 mm. Hg, and a brachial artery systolic pressure of 116 mm. Hg. Brachial artery oxygen saturation was 96 per cent, and dye curves revealed a possible small right to left shunt at the atrial level. Withdrawal tracing from the pulmonary artery to the right ventricle was not technically satisfactory. A diagnosis of valvular pulmonary stenosis with intact ventricular septum and small right to left shunt at the atrial level was made.

At operation on July 22, 1959, the heart was exposed through a midline sternum-splitting incision. There were abnormalities of both coronary arteries with aneurysmal dilations in multiple areas. No palpable thrill was palpable over these coronary vessels. The venae cava were cannulated through the atrium and blood was returned from the modified DeWall-Lillehei bubble oxygenator sigmamotor pump connected to the right femoral artery. Pulmonary arteriotomy revealed a normal pulmonary valve. A finger inserted through the valve revealed diffuse narrowing of the infundibulum. A right infundibulotomy was performed and muscle from the crista removed by sharp scissor dissection. A diamond shaped patch of laminated Ivalon and Teflon was sutured into the ventriculotomy incision in order to enlarge the diffusely narrowed infundibulum. After the cannulae were removed, the pressure in the pulmonary artery rose to 25 mm. Hg, but the right ventricular pressure remained about 100 mm. Hg.

Postoperative course. The patient's postoperative course was complicated by persistent fever for which no cause could be found. He was ultimately discharged from the hospital somewhat improved. The thrill and murmur remained essentially unchanged. After leaving the hospital, the patient felt that his exercise tolerance had increased and his color improved. Although follow-up catheterization has not been carried out,

electrocardiography shows a decrease in right ventricular hypertrophy.

DISCUSSION

The true incidence of infundibular pulmonic stenosis with intact ventricular septum has not been determined. Before 1948, all forms of "pure" pulmonic stenosis were considered very unusual, but since that time, improved clinical and laboratory diagnostic methods have resulted in establishing the diagnosis in an increasing number of patients. It seems likely that pulmonic stenosis with intact ventricular septum comprises about 10 per cent of cases of congenital heart disease, and that of these, approximately 10 per cent are infundibular stenosis.[1, 13] In our small operative series, infundibular stenosis with intact ventricular septum has occurred in 3 of 12 patients with isolated pulmonic stenosis. Swan states that 60 cases of isolated infundibular stenosis have been reported in the literature since 1958.[3, 14]

Keith's explanation that infundibular stenosis occurs because of faulty involution of the bulbus cordis is widely accepted.[8] Acceptance of this thesis does not imply that the pathogenesis is understood. Two pathologic types of infundibular stenosis are described by Lev.[11] In the first type, a fibrotic band is present at the junction of the conus and sinus of the right ventricle which divides the ventricle into two portions. The conus portion is the infundibular chamber. This is the most common variety of infundibular stenosis associated with intact ventricular septum. There may be an associated atrial septal defect or patent foramen ovale. The second type consists of a shrunken conus portion with a long stenotic infundibulum. Johns states that the second type is commonly seen in the transposition complexes.[7] In two of our cases, the infundibular stenosis was quite localized with a well formed infundibular chamber. In our third case, a long area of infundibulum was narrowed, corresponding to the second type described by Lev. Kjellberg has discussed the abnormal positions of the muscle bands comprising the crista supraventricularis which may account for some of the anatomical variation in the isolated infundibular stenosis defect.[10]

CLINICAL FEATURES

The history obtained from patients with infundibular pulmonic stenosis is indistinguishable from that in other forms of isolated pulmonic stenosis. Symptomatology ranges from no symptoms in those patients with mild stenosis through mild limitation of activity and progresses to dyspnea, fatigue, syncope and finally, symptoms of right-sided congestive heart failure. Cyanosis may occur in severe cases in patients who have an associated atrial septal defect or in a patent foramen ovale.[15]

On physical examination, there is usually little or no chest deformity. A left parasternal lift or tap is usually present. The heart may be slightly enlarged. The systolic thrill and murmur are usually maximal in the left second and third interspaces, somewhat lower than in valvular pulmonic stenosis. The second sound is almost always well split and of diminished intensity. It is not well transmitted to the right. The long well known fact that the murmur is maximal at a lower interspace in infundibular than in valvular stenosis was obvious only in retrospect in our patients. Certain phonocardiographic characteristics of the murmur are helpful and will be mentioned subsequently.

DIAGNOSTIC PROCEDURES

Chest x-ray usually shows no increase in transverse diameter of the heart. The right ventricular enlargement is best seen in a direct lateral projection and is frequently marked, although this may be less prominent in infundibular than in valvular stenosis. Of more importance is the fact that the pulmonary artery, which is almost always enlarged because of poststenotic dilation in valvular pulmonic stenosis, is usually of normal size in infundibular pulmonic stenosis. The decrease in size of peripheral pulmonary arteries has been reported to be less marked in the infundibular form of the disease.[10] Absence of an enlarged main pulmonary artery in an x-ray of a patient with the clinical findings of pulmonic stenosis should arouse serious suspicion that the stenotic area is infundibular. Electrocardiography in both valvular and infundibular stenosis shows right ventricular hypertrophy. The degree of right ventricular hypertrophy has been shown to correlate with the height of right ventricular systolic pressure in patients with valvular pulmonic stenosis, and in our experience, this is also true of infundibular pulmonic stenosis.[5] The phonocardiogram may be of help in differentiating isolated valvular

from infundibular pulmonic stenosis. The murmur of infundibular pulmonic stenosis has been reported to start immediately after the first sound and to extend only to the aortic component of the second sound, in contrast to the murmur of valvular pulmonic stenosis which starts later and extends through the sound of aortic closure.[10] Pre- and postoperative phonocardiographic studies in our patients indicate that the difference in characteristics of the murmur depends more on the severity of the stenosis than on the site.

At cardiac catheterization, withdrawal of the cardiac catheter from pulmonary artery to infundibular chamber to sinus portion of right ventricle should result in a pressure tracing diagnostic of infundibular pulmonic stenosis. Typically this tracing reveals no change in systolic pressure on withdrawal from the pulmonary artery into the infundibular chamber, but rather, a sudden drop in diastolic pressure. When the catheter is further withdrawn into the body of the right ventricle, the systolic pressure should increase with relatively little change in diastolic pressure. It was not possible to make the diagnosis of infundibular stenosis in our patients by this technique because technically satisfactory withdrawal tracing could not be obtained. Kirklin has pointed out that even when careful withdrawal tracings are recorded, the diagnosis of infundibular stenosis may be missed.[9] The possibility that catheterization data alone may not prove or disprove a diagnosis of infundibular stenosis is of considerable significance to the surgeon.[2]

The injection of contrast media into the right ventricle with clear visualization of the outflow path is often successful in differentiating infundibular from valvular stenosis. The importance of differentiating infundibular hypertrophy from true infundibular stenosis has been emphasized by Kjellberg.[10] This procedure was not carried out with any of the patients reported herein, but has been of considerable value in subsequent studies on other patients.

SURGICAL TREATMENT

Although attempts have been made to resect the tissue responsible for infundibular stenosis by closed method,[4, 6] there is little question that the direct visual approach allows more accurate and thorough correction. Swan has recommended that direct vision surgery be carried out with the use of inflow occlusion and hypothermia, and has emphasized the minimal risk of ventricular fibrillation with careful hypothermic technique. Resection of the infundibulum has been accomplished in 4 to 5 minutes in Swan's series, and the results have been quite satisfactory.[14] Other surgeons have preferred the use of cardiopulmonary bypass to allow more time for infundibular resection and to prevent the danger of ventricular fibrillation, however slight.[12] In our 3 patients, the heart was bypassed for 19, 26 and 63 minutes, and in two instances, revision of the outflow tract with a plastic patch was considered advisable. The risk of cardiopulmonary bypass has steadily diminished until at the present time there is little hesitation in employing this method if there appears to be a definite advantage gained by its use. The fact that bypass techniques safely allow the intracardiac operating time necessary for extensive reconstruction of the ventricular outflow tract or for repair of unsuspected associated defects has made the approach the procedure of choice at our institution for operations on all cases of pulmonic stenosis except for patients operated on in infancy.

SUMMARY AND CONCLUSIONS

1. Infundibular pulmonic stenosis in the presence of intact ventricular septum is not a rare congenital cardiac anomaly, and probably comprises about 10 per cent of all cases of pulmonic stenosis with intact ventricular septum.

2. In 1959, 3 of 12 patients operated on for isolated pulmonic stenosis at the University of Oklahoma Medical Center have had infundibular stenosis. In none of these patients was the diagnosis of infundibular stenosis definitely established before operation.

3. Important points in preoperative differentiation of this form of pulmonic stenosis from the usual valvular stenosis are as follows: (a) Physical examination reveals a thrill and murmur in the second and third interspaces to the left of the sternum rather than the first and second. (b) Chest x-ray fails to show poststenotic dilation of the pulmonary artery. (c) Cardiac catheterization reveals an area of low systolic pressure proximal to the pulmonic valve. (d) Selective right ventricular angiogram is of definite value in visualizing the site of stenosis.

4. Treatment of this condition by infundibular resection can be accomplished with good clinical

results and a low mortality rate. Because of the time required to carefully repair the right ventricular outflow tract, cardiopulmonary bypass is the method of choice at this center.

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THE USE OF SPLIT THICKNESS SKIN GRAFTS IN THE SURGICAL TREATMENT OF CANCER OF THE MOUTH, HYPOPHARYNX, LARYNX AND CERVICAL ESOPHAGUS*

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The surgical attack on tumors of the oral cavity, pharynx, hypopharynx, and cervical esophagus usually represents a compromise between the necessity to remove adequate margins of tissue around the primary tumor and to leave sufficient tissue to enable satisfactory functional and cosmetic reconstruction. Some of the recurrences which result may be attributed to an overly conservative sacrifice of mucosal margins in an effort to achieve the latter aim. Another theoretical and practical reason for a wide sacrifice of mucosal structures lies in the noted tendency toward the development of multiple tumors in these areas. Most of the cancers in this region originate in the mucosal surfaces, and it must be presumed that whatever influences have operated to induce the neoplastic change in one area may also have produced precancerous changes in mucosal areas immediately surrounding the primary tumor. Such a situation obviously exists in the individual who has multiple areas of leukoplakia in the mouth and has developed one or more squamous cell carcinomata in or adjacent to one of these areas.

It would seem that the surgical approach based on these considerations of mucosal origin and spread, and of multicentricity of origin would demand wide sacrifice of mucosa. Unfortunately, there is no very good source of mucosa to cover defects so created, and one must supply the next best epithelial covering, which is skin. This can be migrated in from a distance with its vascular supply intact, as in skin flaps or tubed pedicles, or supplied only as an epithelial covering, as in the split or full thickness graft. Pedicled grafts have the advantage of not contracting and remaining soft and pliable, but have the disadvantage of requiring multiple stages in their use. Split thickness grafts can be applied at the time of the extirpative surgery and so may eliminate multiple staged procedure. These free grafts have the disadvantage of contracting somewhat and of being less pliable, but in many instances the advantages outweigh the disadvantages.

In individuals who have multiple areas of leukoplakia of the oral cavity with or without definite cancer, skin grafts have long been employed as an epithelial covering to resurface the defect left after removal of the abnormal epithelium. Although full thickness grafts can be expected to take satisfactorily if the area to be covered is not large, split thickness grafts are more suitable if extensive areas of mucosa are removed. The anticipated contracture of the split graft can be overcome to some extent by applying a graft larger in area than the mucosa removed. We have preferred not to employ preformed stents to hold these grafts in place, since the amount of mucosa which must be resected can rarely be estimated with absolute certainty in advance. A satisfactory stent can be composed of gauze saturated in 1 per cent neomycin solution and held in place by sutures from the margins of the graft. Such a stent can be packed in to distend the tissues with more certain approximation of the raw surface of the graft to the underlying tissues and better obliteration of dead space. Use of such a graft may be illustrated by the following case report.

CASE REPORTS

Case 1. Since the age of 10, this 55-year-old white woman had been a heavy user of snuff. Leukoplakia had been present for 6 years, and 2 years prior to admission she had noticed a warty growth in the left lower gum. When examined on March 19, 1959, she was found to have extensive leukoplakia of the alveolar ridges, buccal mucosa and hard palate. There was an infiltrative lesion involving the left buccal mucosa posteriorly and

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the left lower gum and retromolar trigone region. No significantly enlarged nodes were palpable. Roentgenograms of the mandible showed a defect in the left mandible 1 cm. in diameter opposite the angle. On April 13, 1959, the entire left buccal mucosa was excised along with that of the upper and lower gums on the left, the mucosa of the lingual aspect of the lower lip and most of the right buccal mucosa. A marginal resection of the mandible was performed in the region of the left retromolar trigone. The pathologist reported squamous cell carcinoma, grade II, in left buccal mucosa and left lower gum invading the underlying mandible, with multiple areas of carcinoma *in situ* of the remainder of the buccal mucosa and mucosa of the lower lip. Examination 1 year later showed no evidence of recurrence, and satisfactory function of the skin graft. The remaining areas of leukoplakia were unchanged (figs. 1 and 2).

In patients with extensive tumors of the gums, buccal mucosa, retromolar trigone and tonsillar area which are treated by neck dissection and resection of the mandible and primary lesion, closure of the defect left after adequate mucosal excision may be difficult with the mucosa available. The mucosa of the tongue is ordinarily employed in such instances, but the functional result after suture of the tongue to the soft palate or upper gum is frequently not very desirable. Skin grafts may be employed at the time of the primary surgical procedure to give better anatomical position of the tongue and a more satisfactory functional result. These principles may be illustrated by the following two case reports, the second of which shows a very large mucosal defect resulting from removal of a primary infiltrative tumor plus satellite abnormal epithelium.

FIG. 1. Left buccal mucosa and lower gum, showing squamous cell carcinoma and leukoplakia.

FIG. 2. After resection of buccal mucosa and gum and application of skin graft.

Case 2. On November 4, 1958, a 71-year-old white man was seen. He stated that 2 months previously he had developed a swelling of the right cheek which was not relieved by extraction of some teeth in the right lower jaw. There was a huge lesion involving the right lower gum, the retromolar trigone region, anterior tonsillar pillar and the upper gum in the region of the maxillary tuberosity, biopsy of which showed squamous cell carcinoma, grade II. A firm node 1.5 cm. in diameter was present in the right submaxillary area. Roentgenograms of the mandible showed a 4-cm. area of bone destruction. On November 10, 1958, a right radical neck dissection with resection of right mandible, right tonsillar region, pterygoid muscles and posterior end of the right upper alveolar ridge with pterygoid plates was performed. A large skin graft was employed to cover the extensive defect. Pathology report on the excised specimen showed a squamous cell carcinoma, grade II, 6 cm. in greatest diameter, involving the right lower gum and surrounding

FIG. 3. Squamous cell carcinoma of lower gum, buccal mucosa and retromolar trigone.

FIG. 4. Postoperative result after neck dissection, resection of mandible and repair of mucosal defect with skin graft.

tissues with no demonstrable lymph node metastasis. There was no evidence of recurrence when the patient was examined on December 4, 1959, and the cosmetic and functional result was good (figs. 3 and 4).

Case 3. A 52-year-old white man was first seen on March 14, 1957, with a 3 to 4 months' history of soreness of the right posterior mouth, and a 2 months' history of a lump in right neck. A dentist had been consulted at onset of symptoms, but a physician was not seen until a week prior to admission when a biopsy was done. The patient had used chewing tobacco and snuff and had smoked two packages of cigarettes daily. Examination showed an extensive lesion of the right retromolar trigone region, adjacent anterior tonsillar pillar and soft palate, with two early lesions in the left side of the soft palate. A mass 3.5 cm. in diameter was present in the right subdigastric area. On March 21, 1957, a right radical neck dissection with resection of the posterior half of right mandible, retromolar trigone and tonsillar region, and mucosa of soft palate was performed. Pathology report showed squamous cell carcinoma, grade II, of the retromolar trigone region with multiple areas of *in situ* carcinoma of the mucosa of the palate. Metastatic squamous cell carcinoma was present in one lymph node from the subdigastric area. On December 4, 1957, a left radical neck dissection was done for metastatic carcinoma in the subdigastric area. This patient has continued to smoke and has developed other areas of leukoplakia requiring excision. On April 15, 1959, leukoplakia of the posterior pharyngeal wall, and on March 10, 1960, leukoplakia of the left posterolateral pharyngeal wall was resected and skin grafts were applied.

FIG. 5. Esophagram showing extensive lesion of pyriform sinus and upper cervical esophagus.

Cancers of the posterior pharyngeal wall below the level of the soft palate, and cancers of the laryngopharynx and of the cervical esophagus, while not entirely similar as to methods of extension and frequency and routes of metastasis, do pose much the same problem as far as reconstruction is concerned. Except in relatively small lesions of the posterior pharyngeal wall, the surgical procedure necessary to encompass these lesions ordinarily requires laryngectomy with resection of varying amounts of pharynx and esophagus with or without unilateral or bilateral neck dissection. The problem of the defect thus created in the pharynx has been met by numerous ingenious methods which have included the use of local tissue flaps, skin flaps migrated in from a distance, prostheses of foreign material and free skin grafts with some splinting device.[1-7] In an effort to avoid multiple staged procedures, we have been attempting to evaluate the usefulness of the split thickness skin graft in the bridging of these defects. The same principles which have been employed in the application of skin grafts in the mouth have been used in skin grafts in the hypopharynx and cervical esophagus. A preformed stent has not been employed, and the skin graft has been much larger in area than the mucosa resected, to allow for the contracture which is known to take place.

To date, this method of reconstruction has been employed in 24 patients having lesions in posterior pharyngeal wall, laryngopharynx or cervical esophagus. These patients comprise a very heterogeneous group and include 6 patients who represent failure of treatment by other methods—either surgical resection or radiation. As others have found, the one stage reconstruction considerably diminishes the morbidity and improves the morale of the patient. It may be worthwhile even as a palliative procedure, since the expected period of convalescence is short and the rapid re-establishment of normal painless ease of swallowing is much to be desired. Some of the uses and limitations of the method may be illustrated by the following series of cases.

Case 4. A 46-year-old Spanish-American woman was seen on November 11, 1955, with a history of intermittent pain in the throat of 2 years' duration, difficulty in swallowing for 1 year, and 30-pound weight loss. Examination showed a large lesion involving the left pyriform sinus and postcricoid region with multiple nodes in the left neck, the largest being 4 cm. in diameter. Biopsy of the primary lesion was reported to show squamous cell carcinoma, grade II. On November 30, 1955, a left radical neck dissection, thyroidectomy,

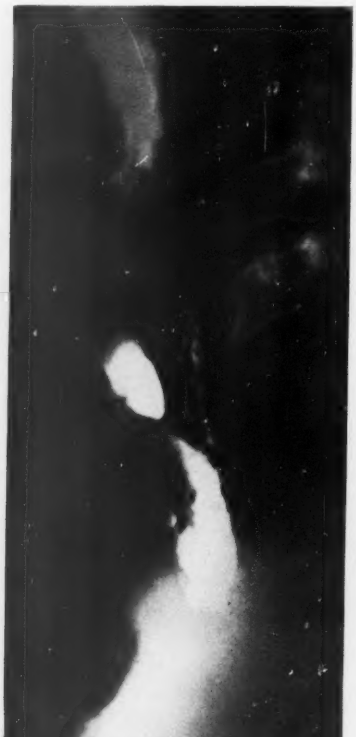

FIG. 6. Esophagus 2 weeks postoperatively

FIG. 7. Esophagus 4 years postoperatively

FIG. 8. Postoperative esophagram showing extensive lesion of pharyngeal wall and pyriform sinus.

FIG. 9. Esophagram 1½ years postoperatively

laryngectomy and partial pharyngectomy and cervical esophagectomy were performed, with immediate reconstruction of the defect with a split thickness skin graft 0.016 in. in thickness. Pathologists reported squamous cell carcinoma, grade II, of larynx and postcricoid region infiltrating the left lobe of the thyroid and sternocleidomastoid muscle, and metastasis in three lymph nodes. The patient was able to swallow satisfactorily on the 10th postoperative day.

FIG. 10. Esophagram showing lesion of posterior pharyngeal wall.

Radiation therapy on the Cobalt 60 irradiator was given from January 4 to February 15, 1956, with a tumor dose of 4600 r. A small fistula was closed on September 25, 1956. The patient has been able to swallow without difficulty, and has even managed a form of bucco-esophageal speech. Examination on March 15, 1960, revealed no evidence of recurrence (figs. 5, 6 and 7).

Case 5. This 71-year-old white woman, who was first seen on January 4, 1956, gave a history of progressive difficulty in swallowing of 1 year's duration, with a 10-pound weight loss. No lesion was seen on mirror laryngoscopy, and there were no masses palpable in the neck. X-ray films of the esophagus showed an almost complete obstruction of the cervical esophagus in the region of C-6-7. A laryngectomy and cervical esophagectomy were performed on January 31, 1956, with re-establishment of esophageal continuity by a split thickness skin graft. Pathology report showed squamous

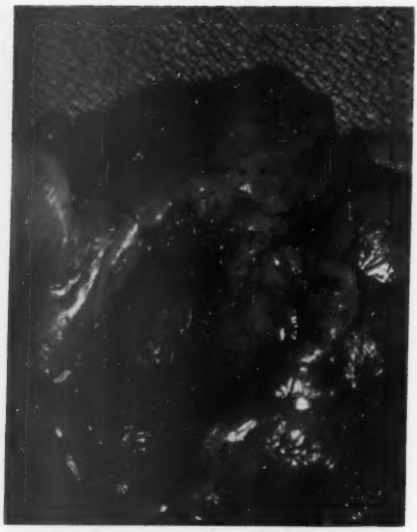

FIG. 11. Operative specimen. Epiglottis is on left margin.

cell carcinoma, grade III, with free margins. Radiation therapy on the Cobalt 60 irradiator was given from February 27 to March 23, 1956, and was stopped because of changes in the skin graft; a tumor dose of 5000 r had been delivered. By July 9, 1956, stenosis of the skin graft had occurred, requiring a staged repair with acromiothoracic skin tube; this was completed in March, 1957. There was no evidence of recurrence as of December 28, 1959.

Case 6. A history of pain in the throat of 3 months' duration, difficulty in swallowing for 2 months, and 35-pound weight loss brought this 59-year-old white man to the hospital on June 30, 1958. Examination showed a very extensive lesion involving the posterior and right lateral pharyngeal wall, the lower tonsillar region and adjacent base of tongue, with questionable fixation to the angle of the right mandible. There were no palpably enlarged lymph nodes. On July 10, 1958, laryngectomy, pharyngectomy, thyroidectomy and marginal resection of the right mandible, with reconstruction of the defect by use of a split thickness skin graft, were performed. The pathology report showed squamous cell carcinoma, grade III, of the pharynx and right pyriform sinus. Postoperative wound healing was rather slow and was complicated by a fistula which developed above the tracheal stoma. On September 19, 1958, swelling and pain developed over the angle of the right mandible, and biopsy showed squamous cell carcinoma. Resection of the right mandible and surrounding tissues was done on October 13, 1958, and a skin graft was again used to close the defect in the mouth. There has been no further recurrence, and when the patient was last seen on February 12, 1960, he was swallowing without difficulty, but had not managed a satisfactory form of speech (figs. 8 and 9).

This method of reconstruction has also been used in a number of patients who had recurrences following either radiation or surgical resection for lesions of the pharynx or larynx. Although the results have not been very good, it is possible to provide rapid reconstruction by this means and thereby, in selected cases, offer some palliation.

Case 7. A 70-year-old white man was first seen on November 2, 1956, because of difficulty in swallowing. A laryngectomy had been done elsewhere 6 months previously. Examination showed a 6-cm. mass in the midline cervical region with almost complete esophageal obstruction. Surgery was refused until December 21, 1956, when obstruction was complete. Resection of upper trachea, cervical esophagus and surrounding tissues was done on December 22, 1956. The pathologists reported that the specimen showed squamous cell carcinoma involving the esophagus, thyroid, trachea, left superior and recurrent laryngeal nerves and the sympathetic trunk. Partial repair of the esophageal defect was done at the time of initial resection by means of a split thickness skin graft, but closure of the pharyngeal stoma was not completed until February 13, 1957. Although the patient underwent several procedures for excision of small areas of recurrent carcinoma in the ensuing months, he was able to swallow without significant difficulty until May 29, 1958, when a gastrostomy was performed. The patient died of recurrence of tumor around the tracheal stoma and in the esophagus and superior mediastinum in June, 1958.

FIG. 12. Operative defect. Uvula is seen projecting beneath base of tongue.

Case 8. At the time that this 52-year-old white man was first seen on July 8, 1959, there was a question as to whether or not radical treatment should be undertaken. He gave a history of having had a sore throat since November 1958, with biopsy of a lesion of the posterior pharyngeal wall on April 6, 1959, followed by radium implant and external radiation. Examination showed an extensive lesion involving the posterior and right lateral pharyngeal walls, with a large mass in the right subdigastric region. Surgical resection on April 15, 1959, consisted of right radical neck dissection, laryngectomy and pharyngectomy, with immediate repair by split thickness skin graft. Pathology report showed squamous cell carcinoma, grade III, of the posterior pharyngeal wall and right pyriform sinus, with metastatic squamous cell carcinoma in five lymph nodes. Healing of the wound was somewhat prolonged, and a minor procedure was required to close a small fistula which developed above the tracheal stoma. The patient, however, was eating satisfactorily 6 weeks after the surgical procedure. On December 6, 1959, a left radical neck dissection was performed, with one metastatic node reported in the operative specimen. When the patient was last seen on March 15, 1960, he was swallowing without difficulty and talking satisfactorily with an electric larynx (figs. 10 through 14).

Case 9. On February 8, 1956, a 58-year-old white woman was first seen with a 6 months' history of difficulty in swallowing, and a 1 month's history of a mass in right side of the neck. She had had a carcinoma of the tongue treated by radium implant 8 years previously. Mirror laryngoscopy showed a large mass in the postcricoid region, and palpation of the neck showed a 3.5-cm. right subdigastric node with a mass in the region of the right lobe of the thyroid. Biopsy of the postcricoid lesion showed squamous cell carcinoma, grade III. It was elected to treat this patient with radiation therapy, and from February 15 to April 11, 1956, the patient received a tumor dose of 7700 r on the Cobalt 60 irradiator. Biopsy of the cervical esophagus on May 10, 1956, showed squamous cell carcinoma, and palpation of the neck revealed persistence of the nodes. It was believed that despite the large amount of radiation received, an attempt at surgical removal should be undertaken. On May 26, 1956, a right radical neck dissection, laryngectomy, thyroidectomy and cervical esophagectomy were performed. The right mediastinal pleura was opened during the surgical procedure. No satisfactory closure using the heavily irradiated skin flaps could be devised; consequently a split thickness skin graft was used to try to close the surgical defect partially. The pathology report showed squamous cell carcinoma of the cervical esophagus and right pyriform sinus with extension to the thyroid gland, and metastatic squamous cell carcinoma in five lymph nodes. For a period of 10 days the skin flaps and skin graft appeared viable, then disintegrated. Death occurred from sudden massive hemorrhage from the carotid or innominate artery.

FIG. 14. Esophagram 6 months postoperatively

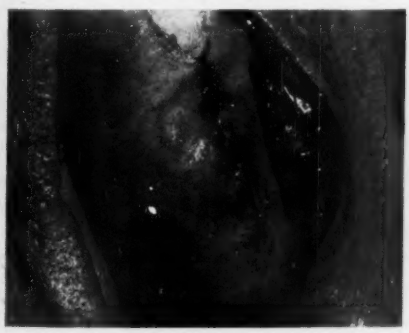

FIG. 13. Skin graft prosthesis

From these experiences, which are limited in number and in years of follow-up, certain tentative conclusions can be drawn. (1) When patients with lesions of the posterior pharyngeal wall are treated surgically, split thickness skin grafts are about the only form of epithelial covering which can be employed. Particularly is this true when the line of resection extends into the nasopharynx, as has been the case in most of the patients we have treated surgically for lesions of the posterior pharyngeal wall. This method may be employed

even after radiation, provided the dose has not been excessive nor the fields too large. (2) When patients with extensive lesions of the larynx and hypopharynx are treated surgically and the resulting mucosal defect cannot be satisfactorily closed as a tube, but there is still a narrow strip of mucosa remaining between pharynx and esophagus, a split thickness skin graft may be employed to bridge the remaining defect with minimal risk of subsequent stenosis. (3) It is probably unwise to irradiate such skin grafts in the postoperative period with the usual therapeutic doses. If irradiation is to be employed, it should be directed to fields along the jugular chain of nodes just lateral to the skin graft tubes. (4) When circumferential defects have occurred in the cervical esophagus or pharynx, immediate reconstruction by a split thickness skin tube is a feasible method, but there is a recognized tendency for these grafts to constrict and for stenoses to occur. This tendency can be minimized to some extent by making the skin tube larger than the original defect, and by arranging the esophageal end of the skin tube so that constriction, if it does occur, will not do so in a circular manner. This can be accomplished by incising the posterior wall of the esophagus and sewing a v-shaped tongue of skin graft into the defect. (5) Split thickness skin grafts may be used in the treatment of postoperative or postradiation recurrences of the larynx involving the cervical esophagus to give a rapid means of re-establishment of esophageal continuity with expectation of palliation. (6) It is unwise to use this method of reconstruction in very heavily irradiated areas if other methods of reconstruction, such as local or distant skin flaps, are feasible.

RESULTS

This group of 24 patients is not a satisfactory one for presenting end results, since it comprises a heterogeneous group of anatomical sites of origin. The group has been used to illustrate principles of mucosal sacrifice and repair. There have been two deaths in the series, one occurring in Case 9, and the other in an elderly woman after surgical resection for mesenteric thrombosis which developed 10 days after an apparently successful removal of a small area of residual or perhaps multicentric tumor on the posterior pharyngeal wall. Of the entire group of 24 patients with lesions of posterior pharyngeal wall, hypopharynx, larynx and cervical esophagus, in whom restoration of continuity was accomplished by means of split thickness skin grafts, 12 are alive without evidence of recurrence from 3 months to 4½ years after operation. In 1 patient, slough of the overlying skin and skin graft was followed by rapid recurrence of tumor; therefore, this re-establishment of esophageal continuity was never accomplished. Two patients have required revision of the esophagus and reconstruction by either local or distant flaps following the development of stenoses at the lower end of the skin tube. The recurrence rate in mucosa has been low, with only 3 patients exhibiting recurrent tumor in the mucosa at the line of resection, a result which could be attributed to too conservative a resection.

SUMMARY

It is believed that a wider sacrifice of mucosa in the surgical treatment of patients with cancer of mouth, hypopharynx, larynx and cervical esophagus might lead to a decrease in the recurrence rate at line of resection. The repair of the defects in mucosa so created may be facilitated by the use of split thickness skin grafts. Methods of use are illustrated by case reports.

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PULMONARY PHYSIOLOGY IN THE EVALUATION AND CARE OF POST-TRAUMATIC PATIENTS*

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Some of the most common and many of the most important complications following general and specific trauma involve the respiratory system. The importance and the severity of such pulmonary complications may be just as great in cases that are not primarily injuries of the chest as they are in primary chest injuries. We feel that a review of normal bronchopulmonary physiologic mechanisms may lead to a better understanding of the changes which take place in the lungs and bronchi following trauma. It is hoped also that such a study will lead to more specific and more effective methods of treatment.

BASIC BRONCHOPULMONARY PHYSIOLOGY

Acute, subacute and chronic changes occur in respiratory mucous membranes, bronchial and bronchiolar walls, peribronchial tissues and in the blood vessels themselves. Many of these changes are characterized merely by transient reversible hyperemia and may not be true abnormalities. On the other hand, inflammation consisting of hyperemia, edema, lymphatic engorgement and stasis, plus the infiltration of leukocytes can also occur.

With healing, resolution may be complete and the tissues may revert to normal, or important residual inflammatory states can remain. The pathologic physiology of such changes may include the following:

1. Bronchial secretions: (a) serous (bronchorrhea), (b) mucoid, (c) purulent exudates.
2. Increased sensitivity of cough reflex.
3. Hyperemia, edema and lymph stasis of mucosa, submucosa, bronchial walls and peribronchial tissues.
4. Leukocyte infiltration.
5. Spasm of smooth musculature of bronchioles.

Any of these changes can cause bronchial obstruction. When several of them are present in the same patient at the same time, the resulting hypoventilation can be profound enough to cause death.

SIGNIFICANCE OF SMALL BRONCHIAL AND BRONCHIOLAR OBSTRUCTION

To many physicians, post-traumatic and post-operative pulmonary complications and the term "atelectasis" mean one and the same thing. Carrying the same thought process farther, "atelectasis" usually connotes a large bronchus plugged with a single large, mucous mass. This condition certainly does occur in this way; however, there is some evidence that obstruction of the smaller bronchi and bronchioles is not only more important than obstruction of the large bronchi, but also that obstruction in the large bronchi may well occur secondary to preliminary obstruction of the small ones. Obstruction of the smaller bronchi by mucus, cellular debris and detritis, mucosal edema, spasm, or a combination of any of these, results in localized faulty pulmonary ventilation. On a small scale, this may present a minor problem. On a large scale, it presents a threat to survival.

CLINICAL EVALUATION OF THE INJURED PATIENT

History. Many injured patients are mentally competent and alert enough to give an accurate history. One should seek evidence of the preexistence of frequent respiratory infections, coughs that persist after a cold, an "asymptomatic cigarette cough," known previous diagnosis of asthma, pulmonary emphysema, bronchi ectasis, recurrent sinus drainage and/or recurrent pneumonitis. Subjective dyspnea and excessive smoking in the history, in addition to any of the abovementioned findings, should suggest subacute, chronic or recurrent inflammation of the respiratory passages, any of which will almost surely predispose the patient to post-traumatic complications.

Physical examination. In addition to the careful physical examination which every injured patient deserves, special attention should be paid to the

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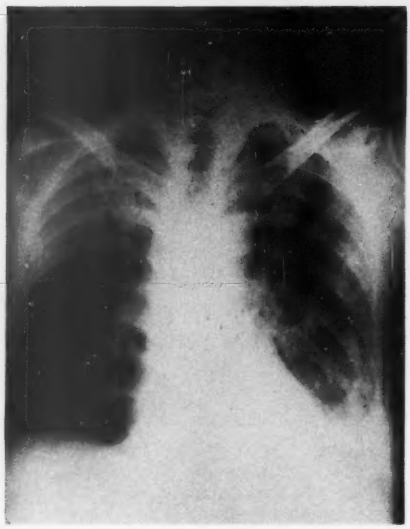

FIG. 1. X-ray of case 1 during life

following points in examining the respiratory system: (1) cough, (2) labored respiration, (3) poor breath sounds by auscultation, (4) inspiratory or expiratory wheezes, rales or rhonchi. Any of these, especially wheezes on expiration, coughing or forced breathing, should alert the examiner to expect pulmonary complications.

DETECTION OF OCCULT HYPOVENTILATION

A high index of suspicion and a careful examination will aid the physician in detecting hypoventilation long before irreversible changes accompanied by cyanosis, delirium or coma occur. There are certain points that can be detected in the history and physical examination which should suggest hypoventilation until proven otherwise. These are: (1) subjective shortness of breath, (2) broken sentence speech, (3) apprehension, obstreperousness, delirium, (4) tachypnea, (5) hypertension due to carbon dioxide retention, (6) skin color changes such as flush or redness, pallor, and cyanosis, especially if accompanied by apprehension, obstreperousness or delirium, in that same order.

CLINICAL EVALUATION *VERSUS* "OBJECTIVE MEASUREMENTS"

Pulmonary function testing, as outlined by Baldwin *et al.*[1] Gaensler,[5] Comroe and associates[3] and many others, has resulted in a striking increase in the knowledge of bronchopulmonary physiology and in the quality of investigative efforts in this field. However, none of the methods or the disciplines employed in this type of work offers the clinician what he needs to evaluate and treat most injured patients. The chief cause of serious and fatal post-traumatic pulmonary complications lies in the realm of the bronchi, the bronchioles, and their physiology. Specifically this paper offers the propositions (1) that a preexisting tendency toward bronchial secretions, bronchial inflammation and bronchiolar spasm is the chief predisposing factor in these complications, one which can actually be predicted, and (2) that small bronchial plugging is the most common cause of failure in airway care. These changes are dynamic. They may not be detected by even the most careful pulmonary function tests.

It is the *tendency* toward these changes which predisposes to pulmonary complications after trauma. If a given patient who has this tendency also has some degree of bronchiolar edema and obstruction at the time that he is injured, he then falls into the category of almost certain pulmonary complications.

CASE REPORTS

We would like to present the following illustrative cases:

Case 1. J. D. B., a 69-year-old white male, was injured on April 18, 1953, when he was thrown from a pick-up truck after a collision, apparently with a bridge abutment. He was hospitalized in a neighboring community and on April 19 was transferred to St. Paul Hospital, Dallas, Texas, under the care of a neurosurgeon. Airway consultation was requested from a thoracic surgeon because of labored breathing. The patient never fully regained consciousness, and a history, as such, was not obtainable from him. The family denied any previous injuries or illnesses, but did state that he had had a chronic "cigarette cough." Physical examination revealed evidence of multiple injuries, including contusions and rib fractures. There were no signs that would suggest localized brain damage. Examination of the lungs revealed no evidence of upper or major airway obstruction and no evidence of pneumothorax or hemothorax. *Wheezes were heard* on the first examination and on every succeeding examination. The wheezes were expiratory and were heard equally throughout both lung fields. They would

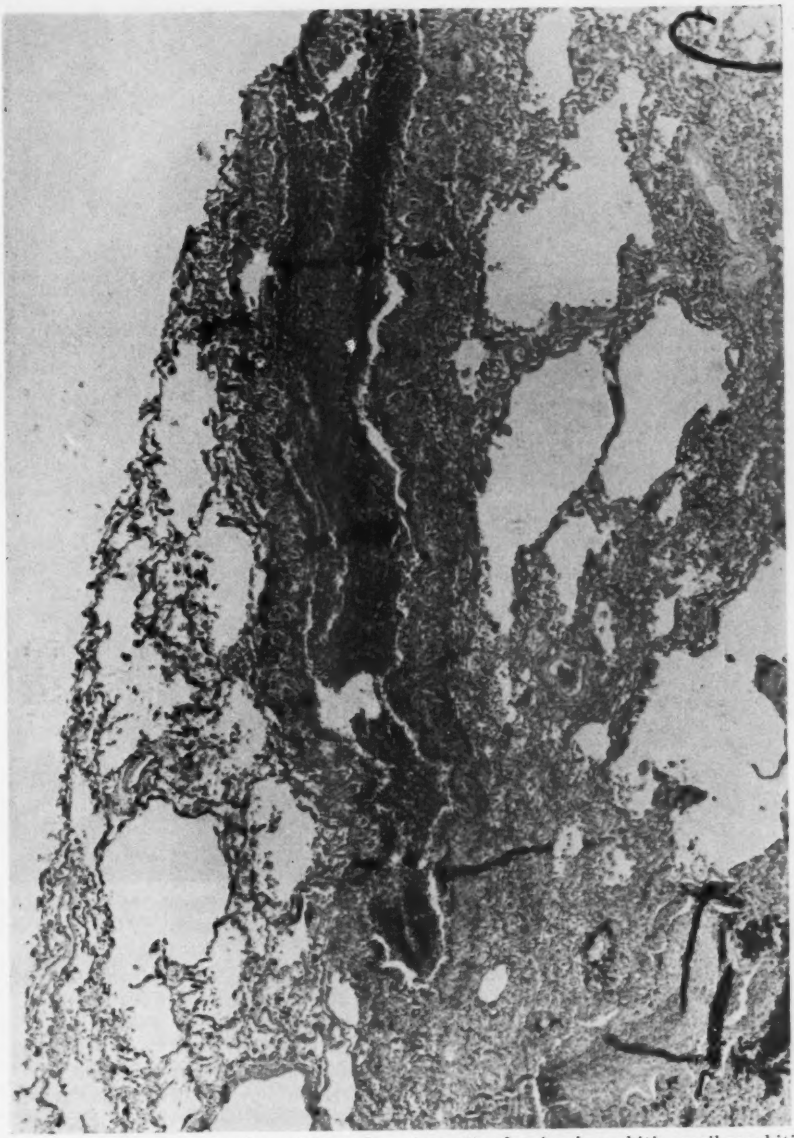

FIG. 2. Low power magnification of bronchus (case 1), showing bronchitis, peribronchitis and a bronchial plug. Hematoxylin and eosin.

clear to a degree after endotracheal suction but never were completely relieved. On April 24, the sixth post-injury day, the patient died suddenly, as if from cardiac arrest.

Figure 1 shows the patient's chest x-ray during life. Figure 2 shows a low magnification of one of the bronchi, showing bronchitis, peribronchitis and a bronchial plug. The third figure is a higher power magnification trichrome stain showing extensive fibrosis in the walls of the bronchiole and almost complete plugging with cellular debris and detritis. Figure 4 points up the extensive chronic inflammation in the wall of the bronchus and the adventitia between the pulmonary arteri-

FIG. 3. High power magnification of bronchus (case 1) showing fibrosis in bronchiole and almost complete plugging with cellular debris and detritis. Trichrome stain.

ole and the bronchiole. This infiltration is with chronic round cells and surely must have preceded by a very long time this patient's injury. Also shown on this section are areas of alveolar rupture and interstitial alveolar thickening.

Case 2. V. B. M. was seen in office consultation on January 21, 1958, because of a cough, wheezing and hemoptysis and a suspicion of neoplasm. He gave a very long history of chronic bronchitis and emphysema. The patient's chief complaint was shortness of breath. His bronchitis and emphysema were characterized by dyspnea, cough, sputum formation and recurrent respiratory infections; a diagnosis of bronchiectasis had been made in the past.

Pulmonary function studies revealed an obstructive ventilatory defect with a 1-sec. vital capacity of 1 L., a ½-sec. vital capacity of 800 ml. and a total vital capacity of 4 L.

Multiple expiratory wheezes were heard on forced expiration, and the breath sounds were distant throughout both lung fields.

Bronchoscopy revealed a tumor growing out of the left upper lobe bronchus. Figure 5 shows a negative posteroanterior roentgenogram of the chest and figure 6 is a lateral view which also failed to show the tumor. Figure 7 is a planogram which shows the tumor in the left main bronchus, at the left upper lobe orifice. Pulmonary complications have the same significance following surgical trauma as they do following external trauma not inflicted by a physician. It was believed that this patient gave ample evidence by his history and physical and pulmonary function examinations

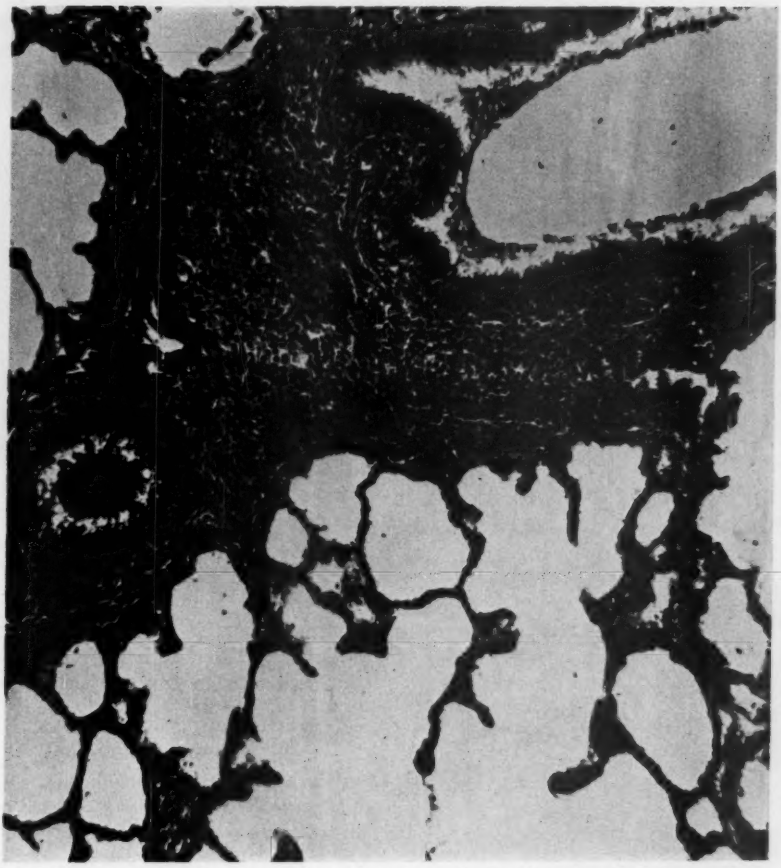

FIG. 4. High power magnification of bronchus (case 1) showing chronic inflammation of bronchus wall and adventitia.

to make a diagnosis of recurrent bronchial edema and secretion formation. For this reason he was considered a certainty for postoperative bronchopulmonary obstruction and hypoventilation, and was prepared very carefully for surgery. He had difficulty breathing for the first 24 hours after pneumonectomy, just as was predicted, but responded well to treatment.

Figure 8 is a photomicrograph of the patient's left lower lobe (which was not obstructed by the tumor) showing emphysema and extensive bronchial thickening. Figure 9 shows the very great chronic inflammation and thickening in the bronchiolar wall; however, the bronchial lumen is clean, because (we feel) of adequate preoperative preparation. Figure 10 is a postoperative chest x-ray following the pneumonectomy. The patient has continued to work and is well at this time. (Added in proof—Since this paper was submitted the patient has died of metastatic carcinoma.)

Comment. In addition to obtaining a history of recurrent bronchopulmonary inflammation, secretions and severe shortness of breath in the second case, we were able to perform objective pulmonary ventilatory function studies. Admittedly, it would be advantageous to perform the same type of tests, either in this simple form or in more detail, upon injured patients, but very often this is not possible for obvious reasons. However, we feel that evidence gained from history and physical examination of recurrent bronchial and bronchiolar inflammation, and the results that ensue, is probably more important than the collection of data on so-called objective

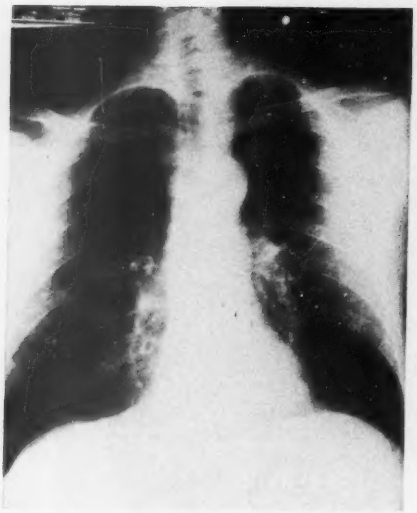

FIG. 5. Case 2, negative posteroanterior roentgenogram of the chest.

FIG. 6. Case 2, lateral view of the chest. Tumor not visible.

pulmonary function testing. When feasible, we recommend that both be done.

Once it has been established, whether by history and physical examination, by bronchopulmonary function testing, or by both, that the patient is likely to develop these complications, preventive measures should be taken.

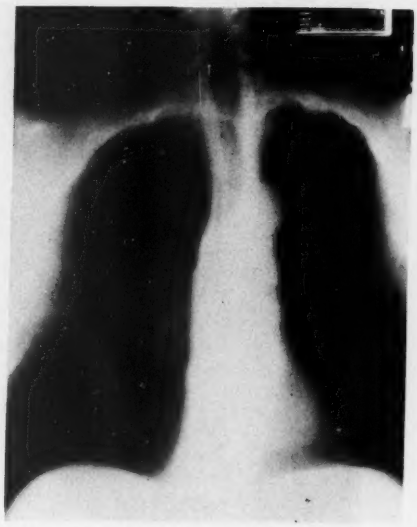

FIG. 7. Planogram of case 2, showing the tumor in the left main bronchus, at the left upper lobe orifice.

PREVENTION OF BRONCHOPULMONARY COMPLICATIONS

Bronchopulmonary complications are best prevented by the prevention of any degree of chilling. A common denominator among practically all patients with reactive and sensitive bronchopulmonary reflexes and membranes is reaction to temperature changes. These reactions represent basic physiologic reflexes, most of which are well understood and have been well documented in the past.[2, 6, 10-15] In simple terms, it can be stated unequivocally that a patient who has a tendency toward bronchitis, bronchiolitis or recurrent respiratory infections will give the following responses to almost any degree of chilling: (1) increased bronchial secretions, (2) hyperemia and edema of the bronchi and bronchioles, (3) tendency toward bronchiolar spasm, (4) sharp increase in the sensitivity of the cough reflex. It is apparent that any or all of these pathologic physiologic changes are detrimental to the patient. They can be prevented by early and adequate application of the following discipline.

1. Do not rely on bed covers or sheets but keep the patient's entire body clothed at all times

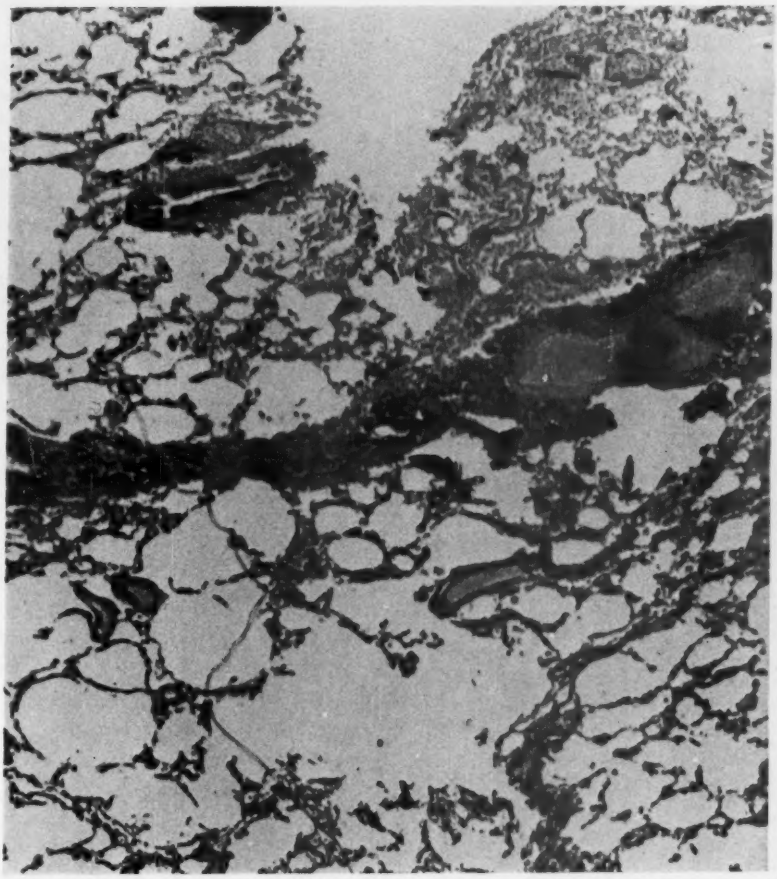

FIG. 8. Photomicrograph of case 2, left lower lobe, showing emphysema and extensive bronchial thickening.

with light weight cotton garments, consisting of long sleeved, long legged cotton pajamas, light socks and a light strap-shouldered undershirt.

2. These garments should be changed as often as necessary to keep them dry.

3. Under all circumstances prolonged exposure to room air, especially in an air conditioned or cooled work area, should be avoided, as for example in the emergency room, x-ray department, recovery room, operating room, during frequent examinations, on carriages, etc. It is necessary to insist that every square centimeter of the patient's skin be covered with light weight cotton garments that are kept dry at all times.

4. If possible, provide the patient with a room of constant temperature, preferably in the mid or upper seventies.

The prophylactic therapy outlined above has proved so effective that we feel that we cannot stress its value too much. It falls to us, however, and to all other doctors, to see patients under circumstances in which prophylactic therapy has not been possible or has not been utilized. Under these conditions restorative therapy is often a matter of life or death.

RESTORATION OF ACUTELY DERANGED BRONCHO-PULMONARY PHYSIOLOGY

Objectives of Treatment

1. Reduce bronchial, bronchiolar and peribronchial edema.

2. Keep the large bronchi and trachea free from mucus.

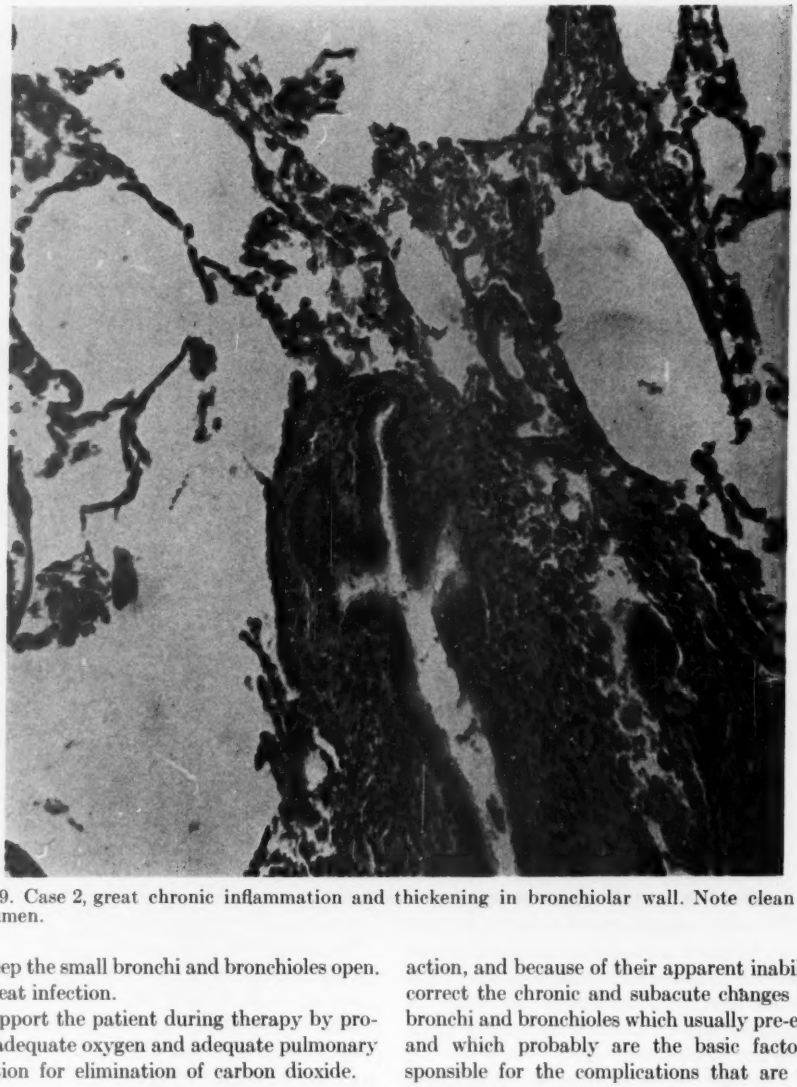

FIG. 9. Case 2, great chronic inflammation and thickening in bronchiolar wall. Note clean bronchial lumen.

3. Keep the small bronchi and bronchioles open.

4. Treat infection.

5. Support the patient during therapy by providing adequate oxygen and adequate pulmonary ventilation for elimination of carbon dioxide.

Methods of Accomplishing This Restoration

1. Infection should be controlled with broad spectrum antibiotics (shotgun).

2. Chemical bronchodilators such as aminophylline, isuprel, aerolone, epinephrin, ephedrine, antihistimines and many others are useful, but their usefulness is limited. (Although we do not object to the use of chemical bronchodilators, we feel that it should be stressed that reliance must not be placed on them because of their short action, and because of their apparent inability to correct the chronic and subacute changes in the bronchi and bronchioles which usually pre-existed and which probably are the basic factors responsible for the complications that are under treatment.)

3. Obstruction of the trachea and larger bronchi should be treated. By far the most effective method and the simplest is endotracheal catheter suction as advocated by Haight[7] and Haight and Ransom.[8]

4. Obstruction of smaller bronchi should be treated. Many times the vigorous cough reflex elicited by an endotracheal catheter will cause sufficient dislodging of the obstructive mucus, detritis and cellular debris in the smaller bronchi

to remove the patient from danger. However, as illustrated in case 1 above, sometimes even very vigorous endotracheal suctioning will not accomplish this. Bronchoscopy is likewise ineffective for small bronchial and bronchiolar obstruction. At the present time it seems that the only way to accomplish release of these tiny mucous plugs is to liquefy them with inspired air containing water in a vaporous state There are many ways in which this might be accomplished; however, in our experience, the only sure way is with a good nebulizer equipped with a warming device. We have used warmed nebulization for the past 10 years with exemplary clinical results. This procedure has been described by Cushing and Miller.[4] Wetting agents may also be used, but these cannot substitute for warmth.

5. Intermittent positive pressure breathing devices may be used. This machine is probably one of the most popular gadgets in the whole field of medicine today. There are many good makes, all of which accomplish about the same purpose. An increase in the pressure during inspiration will provide the patient with a greater partial pressure of oxygen in inspired air. Also an increase of pressure on inspiration, with or without automatic cycling, may provide the patient with enough increase in pulmonary ventilation to rid him of CO_2 and make the difference between success and failure.

Endotracheal suctioning, plus intermittent positive pressure breathing combined with adequate warmed nebulization, with or without bronchodilator drugs, probably represents the utmost in adequate airway care at the present time.

6. Prophylactic measures are important in treatment too. All the measures outlined for prophylaxis are considered very important in treatment and they are recommended just the same as used in prophylaxis.

A LOOK AT SOME COMMON PRACTICES

On the basis of the above outlined physiologic mechanisms, and considering the methods of restoring normal functioning, let us examine some of the common practices in the care of injured patients today. There are three very common practices in the care of post-traumatic and postsurgical patients which are worthy of mention, the first of which is the use of narcotics. Lasagna and Beecher[9] and many others have pointed out that small doses of narcotics are just as effective and far safer than large doses. In spite of strenuous efforts to warn against the overuse of narcotics, injured and postoperative patients are still receiving very dangerous dosage schedules, and this practice is quite widespread. In a given patient who has airway obstruction resulting in hypoventilation, the use of narcotics can result only in further decrease in pulmonary ventilation, less effective cough and a worsening of the airway obstruction. The most grave danger occurs when mental changes such as apprehension, restlessness or delirium, which are caused by hypoventilation in the first place, are treated with narcotics or sedatives, further compounding the physiologic derangement.

FIG. 10. Postoperative chest x-ray of case 2, following pneumonectomy.

A second common practice in postinjury and postsurgical situations is nakedness. There are times when nakedness is either unavoidable or desirable. However, we wish to stress the fact that exposure of the skin of the arms, legs, shoulders or the body to room temperature in the normal work area might be very detrimental to any patient, and is specifically contraindicated in patients with pre-existing bronchopulmonary problems.

A third common practice is the use of a refrigerated oxygen tent which dehumidifies and cools the patient and his inspired air.

CONCLUSIONS

1. Post-traumatic and postsurgical bronchopulmonary complications can be predicted, pre-

vented and treated on the basis of a careful clinical examination and physiologic therapy.

2. The routine use of narcotics in "normal" dosage schedules may be very detrimental and should be condemned.

3. Exposure of any portion of the patient's body to normal work area temperatures can compound derangements in pulmonary physiology and can cause or aggravate bronchial obstruction and a hypoventilatory state.

SUMMARY

An attempt has been made to review the normal bronchopulmonary changes and one of the basic causes of acute, subacute and chronic inflammation. Special emphasis has been placed upon the significance of small bronchial and bronchiolar obstruction. Methods have been outlined by which such states can be detected clinically. Two illustrative case reports have been presented, one of an injured man who died of widespread bronchiolar obstruction and one of a surgical patient who might have died had not prophylactic and therapeutic measures been used as outlined. A discipline has been outlined which is aimed at preventing the bronchial and pulmonary changes which result in hypoventilation, and a method of treatment has been suggested for the restoration of acutely deranged bronchopulmonary physiology.

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GASTRIC CARCINOMA*

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Gastric carcinoma, which until recently was the most frequent visceral cancer encountered in man, now occupies third place, having been superseded by cancer of the lung and cancer of the colon. This is due to two factors; namely, (1) the incidence of cancer of the lung is increasing more than that of any other cancer in the body, and when both sexes are considered, the incidence is even higher than cancer of the breast, and (2) the incidence of gastric cancer is decreasing. From 1947 to 1957, deaths from gastric cancer in the United States decreased from 18.1 per 100,000 population to 12.6. Helsingen and Hillestad[26] reported similar observations in Norway, where the death rate from gastric cancer has steadily decreased since 1920.

It is interesting to speculate about why gastric cancer is becoming less common, because with increasing longevity cancer usually becomes more prevalent, since it is primarily a disease of older persons and the incidence in people older than 40 years increases with each decade. One would therefore expect the incidence of cancer of the stomach to increase rather than decrease.

Although the curability rate of gastric cancer has increased slightly, this increase is not enough to account for the decrease in the death rate in the past 10 years. There are two possible factors responsible for this decreased death rate: first, the performance of gastrectomy for lesions that are not clinically malignant, such as gastric ulcer, but which prove histologically to be cancer; and second, the removal of premalignant gastric lesions, such as chronic gastric ulcer, polyps and certain cases of chronic gastritis. Although most authorities now are of the opinion that gastric cancer seldom if ever develops in a pre-existing benign ulcer but begins *de novo*, it is our belief that malignant change in a benign gastric ulcer is possible and probably occurs more often than is commonly believed.

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Malignant lesions of the stomach occurred once in every 200 patients admitted to the Mayo Clinic,[6, 57] and at Hines Veterans Hospital, 0.6 per cent of all patients admitted and 4.7 per cent of all cancer patients had cancer of the stomach.[33] One of every 681 patients admitted to the Ochsner Clinic had gastric carcinoma. The incidence of gastric cancer apparently varies widely in different countries. It is extremely high in Japan and in Iceland, where 50 per cent of all malignant lesions in males occur in the stomach. Because smoked foods are eaten in large quantities in Iceland, Dungal[18] suggested that this may be an etiologic factor.

ETIOLOGY

The cause of gastric carcinoma is not known, but certain lesions are presumably precancerous. For example, certain cases of gastritis appear to be premalignant. Konjetzny[30] believed that approximately 85 per cent of gastric cancers develop from pre-existing chronic gastritis. Orator,[43] Hurst[28] and Puchert[45] held similar views. Individuals with pernicious anemia and associated chronic atrophic gastritis have a higher incidence of carcinoma of the stomach than others. Among 1320 patients with pernicious anemia treated at the University of Michigan Hospital, Rubin and Hodges[46] found 35 with gastric cancer (2.5 per cent); this was 10 times the incidence in the general hospital population. They emphasized the fact that the malignant lesion occurring in patients with pernicious anemia is usually in the fundus or body of the stomach, and they believed that the gastritis is definitely precancerous. Schell, Dockerty and Comfort,[49] who analyzed 94 cases of gastric carcinoma in patients with pernicious anemia, reported similar observations. They believed that carcinoma associated with pernicious anemia is different from that seen in other individuals. It tends to be polypoid and usually is multicentric, located in the fundic or cardiac areas, and of a lower grade, frequently has a mixture of grades, and becomes symptomatic relatively late. In a careful study of 69 patients with pernicious

anemia, Siurala and associates[52] found that the incidence of gastric cancer was 7.5 per cent and was 9.8 per cent in those older than 60 years. This is considerably higher than the percentage in the general population in Sweden, which is 0.15 per cent in patients older than 40 years and 0.34 per cent in those older than 60 years; thus the incidence is 29 times greater in patients older than 60 years with pernicious anemia than in the general population. They also found that cancer of the stomach was 24 times as frequent among the relatives of patients with pernicious anemia as it was among the relatives of the controls. The mean duration of the pernicious anemia was twice as long in the group with carcinoma as it was in those without carcinoma. The incidence of gastric carcinoma and gastric polyps in the patients with pernicious anemia was 7.5 per cent and 6 per cent, respectively.

In an extensive study of stomachs removed for ulcer or carcinoma, Warren and Meissner[50] concluded that there are two types of gastritis: (1) exudative and (2) epithelial. In the latter, epithelial changes, consisting of excessive formation of mucus, decrease in the chief and parietal cells and the formation of atypical gastric glands which appear embryonic with increased mitoces, are precancerous and may resemble the changes in the cervix, skin and breast. On the other hand, the exudative changes are inflammatory, reversible and not precancerous.

Recently much has been published concerning development of carcinoma in the residual gastric pouch after gastroenterostomy or resection for benign gastric and duodenal disease. Singer[51] stated that after gastrojejunostomy there develops in the region of the stoma, which is subjected to regurgitation of alkaline intestinal juices, atrophic or hypertrophic gastritis with premalignant polypoid changes. Kühlmayer and Rokitansky[32] found that at necropsy the incidence of cancer of the stomach in patients not previously operated upon was 5.9 per cent, as compared with an incidence of 10.6 per cent among 363 patients who had had previous gastric resections, and 12 9 per cent after gastroenterostomy for benign disease. In the age group from 40 to 50 years, the percentage of carcinoma in the stump was 21 as compared with 7 in the group not operated upon. Kühlmayer and Rokitansky emphasized that the malignant lesion developing after a previous operation offers a better prognosis, since 82 per cent of their patients with lesions in the gastric stump lived from 5 to 25 years postoperatively and only 6 per cent lived for less than 5 years; 12 per cent lived for more than 25 years.

In 1954, Freedman and Berne[19] collected 58 cases of carcinoma developing in the gastric pouch after gastric operations for benign disease and reported 3 of their own. Helsingen and Hillestad[26] found that the incidence of gastric cancer was higher after resection for gastric ulcer than for duodenal ulcer and the incidence of cancer in the latter was no greater than that in the normal population. However, the incidence was 3 times greater in the patients who had had resection for gastric ulcer than it was in the general population. In the resections for gastric ulcer, 10 carcinomas were observed, whereas the expected incidence was 3.8. Among patients who had resection for duodenal ulcer, carcinoma developed in only 1, whereas the expected incidence was 1.81. Similarly, Côté and associates[13] believed that the incidence of gastric cancer is greater after resection for gastric ulcer than for duodenal ulcer and that usually the lesion is of a lower grade malignancy than that in patients not previously operated upon. The lesions in 6 of their 17 patients were graded as 2. Of the 17 patients, 12 had had resection for gastric and 5 for duodenal ulcer.

On the other hand, DeBray and associates[15] believed that the prognosis in patients with carcinoma developing in the postgastrectomy gastric pouch is poor. This is possibly due to the fact that the diagnosis is frequently made late because the true condition is not suspected. Krause[31] found no difference in the incidence of cancer developing in the residual gastric pouch after operation for duodenal and gastric ulcers. Of 361 patients with high resection and Billroth II anastomosis, cancer developed in the gastric pouches of 25 (11.3 per cent). Krause also believed that the gastritis resulting from regurgitation of the duodenal content is the factor responsible. Gremmel and associates[22] reported that during a period of 10 years, of 1709 patients having gastric operations for benign lesions performed from 6 to 13 years previously, cancer developed in 13 (11 per cent). They believed that this is a much higher incidence than would occur in the general population. They, too, were of the opinion that gastritis is an etiologic factor. Other cases have been reported.[3, 5, 7, 11]

Colizzi and Servadio[11] emphasized that the prognosis is poor because of the low resectability incidence, which in his series was only 50 per cent.

There is considerable difference of opinion as to whether pre-existing benign gastric ulcers ever become malignant. In 1883, Hauser[24] demonstrated that malignant change could occur in gastric ulcer. Rosenheim[47] in 1890 and Hayem[25] in 1895 also called attention to this possibility. Wilson and MacCarty[62] in 1909 found evidence of gastric cancer developing on the basis of pre-existing ulcer in 71 per cent of 153 cases of proved gastric carcinoma. Among 82 patients who had gastrectomies for cancer, Stout[54] found evidence of cancer developing at the margin of the pre-existing ulcer in 13.4 per cent, and among 138 patients subjected to radical operation, Ekström[18a] found that cancer originated in an ulcer in 16 (11.5 per cent). In spite of this evidence, many believe that there is no relationship between the pre-existing ulcer and cancer. In a study of 375 cases of gastric cancer, Swynnerton and Truelove[55] reported that 27 (7 per cent) of the lesions arose in the gastric ulcer. Deloyers and Moyson[16] stated that 10 per cent of gastric ulcers become malignant. Hess[27] believed the incidence to be 8 per cent, but believed that only approximately half were recognized as malignant. Brown, Fisher and Hazard[9] found only 8 instances (1.1 per cent) of gastric carcinoma apparently developing in the 715 cases of gastric ulcer at the Cleveland Clinic. Urban[57] found evidence of cancer developing in the edge of a pre-existing benign ulcer in 5.9 per cent of 100 resected ulcers. All of these were in ulcers 2 cm. or more in diameter. Gordet[21] found only 7 cases of cancer which developed in pre-existing gastric ulcers in 10 years in the Gastroenterological Department at the Salpetriere Hospital in Paris. Mallory[36] believed that gastric cancer begins in lesions which are carcinoma *in situ*, and that in most instances when an ulcer and cancer coexist, the lesion consists of ulceration of the malignant lesion and not development of cancer in a previously benign lesion.

Gastric adenomas are definitely premalignant. In a study of 195 cases of adenomatous polyps of the stomach, Cromer and associates[14] found that 35 (20 per cent) were malignant. Berg[4] reported that among 106 gastric adenomas from 45 patients, 13 per cent had transition to invasive cancer, 65 per cent had some evidence of cellular atypia and 95 per cent showed more than one focus of proliferation in the stomach; he deduced that they were either multiple neoplasms or other foci of epithelial hyperplasia. He concluded "that gastric adenomatous polyps may be precancerous lesions and also that they are reflections of generalized gastric neoplastic tendencies."

SURVIVAL RATES

Despite the fact that the incidence of gastric cancer is decreasing, it is still a formidable disease because the end results of its treatment are not good. The 5-year survival rates reported from recognized medical institutions have varied from about 5 per cent[10, 33] to 14 per cent.[6, 39, 48, 53, 60] Even more recently, in spite of all the advances that have been made, the results remain poor. In 1958, McNeer and associates[38] reported a 7.1 per cent 5-year survival rate in 1623 patients with gastric cancer who were admitted to the Memorial Hospital. Lehmann and associates[34] had a 9 per cent survival rate. Among 1708 patients with gastric cancer seen at the Lahey Clinic, the 5-year survival rate from 1932 to 1939 was 5 per cent and from 1950 to 1953, 14.7 per cent[37]. Of the patients in our series, 27 (10 per cent) survived for 5 years. Similar results are found in other countries as well. Andreassen[1] reported a 3-year survival rate of 9 per cent of 451 patients treated at the University of Copenhagen Hospital. Similarly, Denck and Helmer[17], in a series of 1429 patients admitted to the University of Vienna Hospital, reported a 5-year survival rate of 8.5 per cent. Levrat and Lesbre[35], in a series of 426 cases from Lyon, reported a 3-year survival rate of 14 per cent and a 5-year survival rate of 7.2 per cent.

It is thus seen that the outlook in gastric carcinoma remains bad, despite the fact that it is one of the oldest diseases known, and despite the tremendous advances that have been made in technical surgery. For some time we[40-42] have maintained that the therapeutic results of gastric cancer can be improved not by more radical operations, such as total gastrectomy in all cases of gastric cancer, but only by operating on patients early in the course of the disease. As previously emphasized, we believe that wide excision of the stomach, first portion of the duodenum, *en bloc* removal of regional lymph nodes and splenectomy are necessary. Denck and Helmer[17] emphasized the necessity of wide resection (more than 3 cm. beyond histologic evidence of tumor). Their 5-year survival rate after radical resection was

32.5 per cent, but only 19 per cent after limited resection. Fretheim[20] demonstrated, however, that total gastrectomy (abdominothoracic) does not necessarily increase the 5-year survival rate. He obtained an 11.1 per cent 5-year survival rate after total gastrectomy and 22 per cent after subtotal abdominal gastrectomy.

Unfortunately, it is difficult to make a diagnosis of gastric cancer while the lesion is still limited to the stomach. This is the reason that we have stated for some time that if results are to be improved, operation should be performed before a diagnosis of cancer has been made. Although this seems paradoxical, it means that lesions not clinically diagnosable as cancer (such as gastric ulcer, gastric polyps, certain cases of chronic gastritis, particularly those associated with pernicious anemia) and rarely, previously well individuals in whom intractable gastric symptoms develop should be given the benefit of abdominal exploration.

Unfortunately, in a large number of patients, operation is obviously contraindicated because of extensive spread at the time that the patient is first seen. Evidences of inoperability are palpable supraclavicular nodes due to malignant cells being carried through the thoracic duct to the supraclavicular fossa, the presence of a rectal shelf resulting from peritoneal seeding, and obvious hepatic metastases. Although it is generally considered that the earlier the diagnosis is made and therapy instituted, the better the prognosis, this is not necessarily the case, because as Andreassen[1] has shown, frequently patients with longer histories of carcinoma have a better prognosis. Of 13 patients with symptoms of less than 1 month's duration, only 1 survived for 3 years, whereas of 120 patients with symptoms of more than 1 year's duration, 26 per cent survived for 3 years. Similarly, Denck and Helmer[17] observed that patients with a short history of symptoms have a poorer prognosis than those with a long history. In our series, the length of history had little effect on the resectability incidence but did affect the survival rates. Of those patients with symptoms of less than 9 months' duration, the resectability incidence was 38 per cent as compared with 44 per cent in those with symptoms of longer than 9 months' duration. The 5-year survival incidence was 7 per cent in those with symptoms of less than 9 months and 16 per cent in those with symptoms of more than 9 months' duration (fig. 1). Denck and Helmer[17] similarly found that the duration of symptoms had no influence on resectability but did affect the ultimate survival rates. McNeer and associates[38] reported a lower resectability rate in those patients with short histories of symptoms. Thus, in patients with long histories, the original symptoms were due either to a pre-existing ulcer which underwent malignant change or to a slowly growing carcinoma. Everything else being equal, however, the earlier the diagnosis is made, the better the prognosis. According to Denck and Helmer[17], the resectability incidence varies with

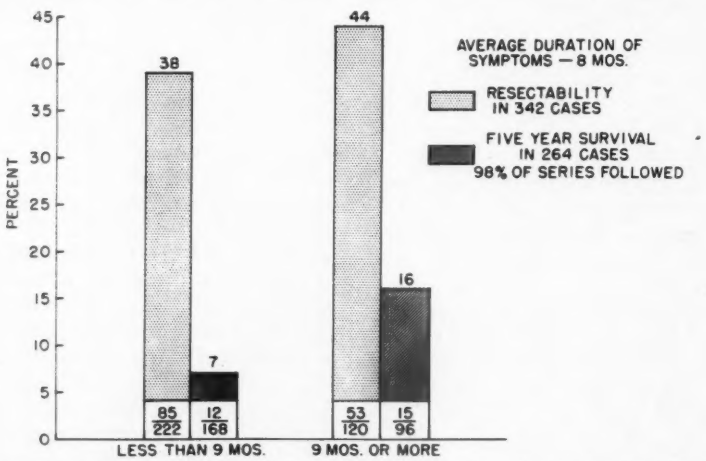

FIG. 1. Comparison of resectability and 5-year survival rates according to duration of symptoms in 264 patients with carcinoma of the stomach.

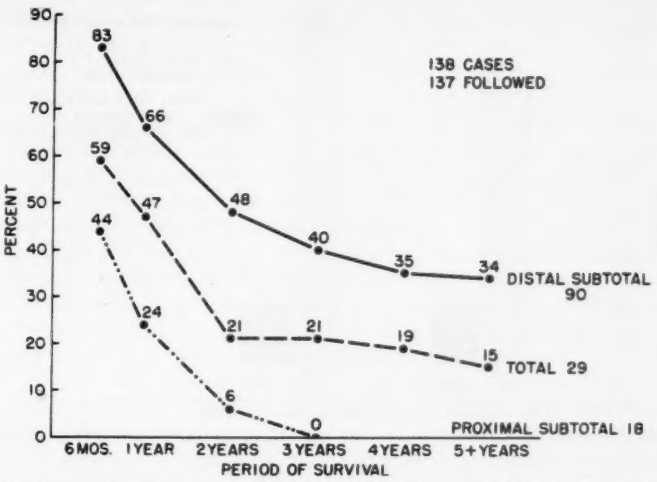

FIG. 2. Survival according to type of resection in 138 patients with carcinoma of the stomach

the location of the lesion. In 1429 cases they noted that resection was possible in 43.2 per cent. Resection was possible in 65 per cent of those in the antrum, in 44 per cent of those in the body and in only 39 per cent of those located high in the stomach.

Andreassen[1] reported a 3-year survival rate after resection of 22 per cent in 72 patients with pyloric lesions, 25 per cent of 110 patients with lesions in the body and none in 29 patients with cardia lesions. The location of the lesion greatly influenced the prognosis in our cases. Of those patients with proximal lesions requiring proximal subtotal gastrectomy, none survived for 3 years. Of those with diffuse lesions requiring total gastrectomy, 15 per cent survived for 5 years, and of those with distal lesions treated by distal radical subtotal gastrectomy, 34 per cent survived for 5 years (fig. 2).

Denck and Helmer[17] also noted that the age had no effect on the operability rate, but that it definitely influenced the prognosis. The 5-year survival rates in those with gastric resections were as follows: from 20 to 40 years of age, 45 per cent; from 40 to 60 years, 31 per cent and older than 60 years, 21 per cent. In our cases the resectability incidence in patients younger than 60 years of age was slightly lower (33 per cent) than in those older than 60 (40 per cent). However, the 5-year survival incidence was the same in those younger as in those older than 60 (fig. 3). This slight difference may be due to the fact that the normal life expectancy is less in individuals older than 60. McNeer and associates[38] also found that the resectability incidence was less in young people.

Kolosova[29] reported that in a series of 4952 patients with gastric carcinoma seen in Russia, a radical operation could be performed in only 15.4 per cent. The resectability incidence was 31.6 per cent of those admitted to the hospital and 44.3 per cent of those operated upon. Lehmann and Morrison[34] reported an operability incidence of 88 per cent and a resectability incidence of 58 per cent among 393 patients admitted to the Wayne University Hospital. Resection with some hope of cure was done in 37 per cent. McNeer and associates[38] reported that of 1623 patients with gastric cancer seen at the Memorial Clinic from 1931 to 1955, 81 per cent were operated upon, of which 33.9 per cent were definitely resectable. Their operability and survival rates showed an increase in the more recent years. From 1931 to 1940, the operability incidence was 65.8 per cent and the 5-year survival rate 3.6 per cent, whereas from 1941 to 1950, the former was 90.8 per cent and the latter 9.1 per cent. Of those patients in whom resection could be done, 27 per cent survived for 5 years. Polypoid and ulcerative lesions gave the best prognosis, 44.8 per cent and 45.5 per cent, respectively. Of our 342 patients, 300 (87.7 per cent) were considered to have operable lesions, of whom 26 (7.6 per cent of total) refused operation and 274 (80 per cent of total) had exploratory operations; 138 (40.4 per cent of total) had re-

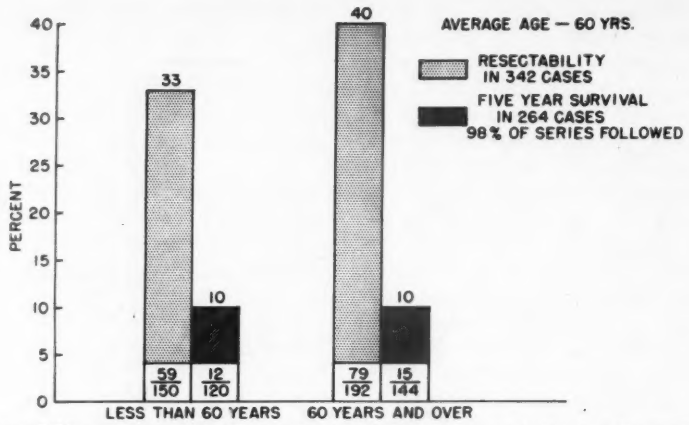

FIG. 3. Resectability and 5-year survival rates in relation to age of patients with carcinoma of the stomach.

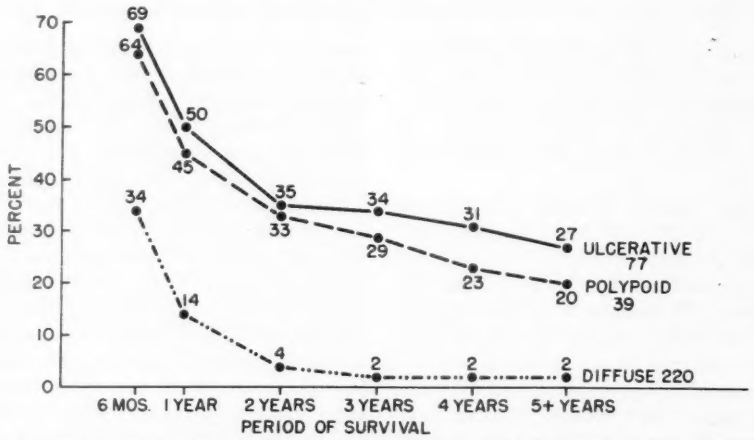

FIG. 4. Comparison of survival rates according to gross pathologic type of tumor in 336 patients with carcinoma of the stomach who were followed (98 per cent of series).

sections. Moore and Mortin[39] reported 12 (14.6 per cent) 5-year survivors among 82 patients with polypoid lesions. Pack and McNeer[44] reported that of patients with polypoid gastric cancer, 23.6 per cent survived for 5 years. Of Moore and Mortin's 57 patients with ulcerative lesions, 28 per cent survived for 5 years and 33.9 per cent of Pack's patients with ulcerative gastric carcinoma survived for 5 years. According to Denck and Helmer[17], polypoid lesions offered the best prognosis. In a series of 226 gastric cancers which clinically simulated gastric ulcer and which represented 25 per cent of all resections for gastric cancer, Comfort and associates[12] reported a lower hospital mortality rate (approximately the same as for benign ulcer) and a higher 5-year survival than for other gastric cancers. In our series the results were best in ulcerative lesions (27 per cent 5-year survivals), followed by polypoid lesions (20 per cent), and the prognosis was worst in diffuse lesions (2 per cent) (fig. 4).

The presence or absence of lymph node involvement also affects prognosis. Denck and Helmer[17] reported that the 5-year survival rate in patients with lymph node metastasis was 14.8 per cent and in those with no nodal involvement, 44 per cent. Welch and Wilkins[61] reported a 5-year survival rate of 63 per cent when the lymph nodes were not

involved at the time of resection. Hammond[23] reported that in Connecticut the 5-year survival rate in those patients with lymph node involvement was 11 per cent, and 48 per cent in those with no nodal involvement. In our cases, extension to the lymph nodes greatly influenced the prognosis. Of 71 patients with nodal metastases who were operated on more than 5 years ago, only 9 are alive (13 per cent), whereas of 31 without nodal metastases, 18 (58 per cent) are alive more than 5 years after resection. It is of interest that within the past 3 years we have seen no patients without lymph node involvement. Pack and McNeer[44] obtained a 5-year survival rate of 24 per cent in those with nodal involvement and 42.8 per cent in those without nodal involvement. In Safar and Cliffton's[48] series, 16.6 per cent of those patients with nodal involvement survived for 5 years as compared with 71 per cent of those without nodal involvement. Moore and Morton[39] stated that lymph node metastasis varies according to the type of gastric lesion. In 118 cases of papillary carcinoma, lymph node metastases were present in 44 per cent, in 82 cases with ulcerative lesions, 41 per cent, and in 31 cases with local infiltrating lesions, 80.6 per cent. In only 5 of 12 cases of linitis plastica (41 per cent) was there lymph node involvement. On the other hand, Black and associates[8] noted that lymph node metastases had little effect on long term survival, but that rather, the prognosis depended upon the degree of lymphoid infiltration in the primary tumor, on sinus histiocytic changes and on the degree of nuclear differentiation in the regional lymph nodes. They found lymph node metastases in more than 40 per cent of the 5-year survivors and no evidence of lymph node metastases in 30 per cent of those dying within 2 years postoperatively.

In a series of 637 patients admitted to the Massachusett General Hospital from 1947 to 1956, Welch and Wilkins[61] performed palliative resections in 153 and operations for cure in 274. They considered the type of tumor to be the most important factor in prognosis, but also emphasized the importance of early diagnosis, particularly in patients with benign ulcer, polyps and pernicious anemia.

Arhelger and associates[2] emphasized the importance of metastasis to the nodes of the hepatic pedicle. Of 26 patients in whom a second-look operation was performed after gastric resection for carcinoma, residual cancer was found in the lymph nodes, fibrous tissue or fat of the hepatic pedicle in 11. Of 24 patients treated by an extended operation, including removal of these nodes, 11 had metastases to the nodes in the hepatic pedicle. This was particularly true in distally located lesions. In 9 of the 11 with hepatic pedicle involvement, the lesion was limited to the pylorus or body of the lesser curvature. Andreassen[1] found metastasis to the hepatic pedicle of the stomach in 12 of 30 patients with carcinoma of the pylorus or body, and also emphasized the admonition of Arhelger and associates that these nodes should be removed at the original operation. In 36 surgical specimens after resection of the stomach and gastric cardia for cancer, Šerý and Dvořáček[50] found 3258 lymph nodes, of which 257 (10.9 per cent) contained metastasis. Of the left gastric nodes 14.1 per cent were metastatic, of the hepatic nodes, 11.3 per cent, and of the lienal nodes, 4.3 per cent. Of the 63 examined, 23 had no metastasis while 40 had metastasis. Of the 40 with metastasis, the positive node involvement was as follows: left gastric, 20.2 per cent; hepatic 17.9 per cent; and lienal 6.7 per cent. The lienal involvement varied according to the location of the tumor. Carcinoma of the esophagocardiac portion and upper third of the stomach had lienal involvement in 5.4 per cent. Those carcinomas involving the middle third (body of stomach) had 18 per cent, those in the pylorus and antrum 1.2 per cent, and those involving the entire stomach, 50 per cent. Šerý emphasized the necessity of including splenectomy in the radical treatment of carcinoma of the stomach.

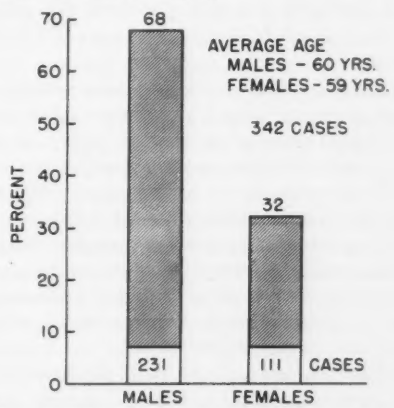

FIG. 5. Distribution of 342 patients with carcinoma of the stomach according to sex.

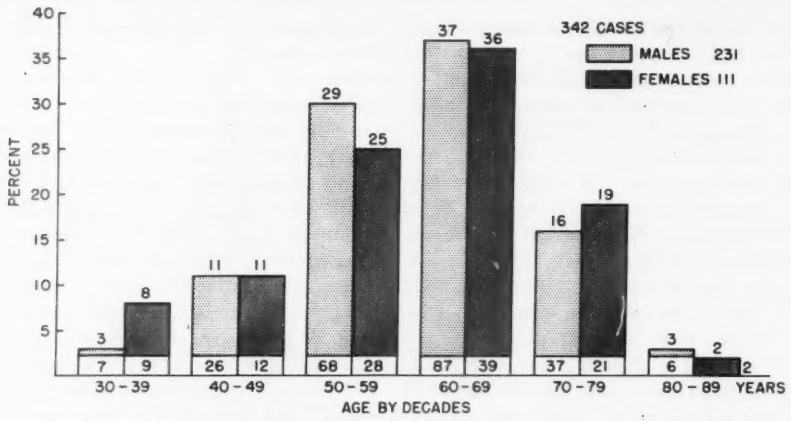

FIG. 6. Distribution of 342 patients with carcinoma of the stomach according to decade of life

ANALYSIS OF PERSONAL SERIES

At the Ochsner Clinic from January 1, 1942 to January 1, 1960, there were 342 cases of proved gastric carcinoma, of which 231 (68 per cent) were men and 111 (32 per cent) were women (fig. 5). The average age of the men was 60 years and of the women, 59. The highest incidence was in the seventh decade, followed in frequency by the sixth and the eighth (fig. 6).

The average duration of symptoms was 8 months, and was slightly longer in men (8.7 months) than in women (7.9 months). The principal symptoms were pain (81.3 per cent), nausea (59.3 per cent), anorexia (57.3 per cent) and dysphagia (18 per cent) (fig. 7). Although anorexia was a complaint of only slightly more than half our patients at the time that they were first seen, we believe that it is one of the earliest manifestations of gastric cancer, one which is frequently disregarded and then becomes overshadowed by later and more severe symptoms. When it appears early, it is of extreme significance and should never be disregarded. In fact, we believe that abdominal exploration is justified in a man who previously has had no gastric symptoms but who, in spite of therapy, complains of anorexia which is severe and persistent enough to cause a weight loss of 15 or more pounds, even though all laboratory studies fail to disclose any abnormality. In such cases, we have demonstrated a small resectable gastric cancer.

Of the physical findings, loss of weight is by far the most important, having been present in 87 per cent of our cases. There was an average loss

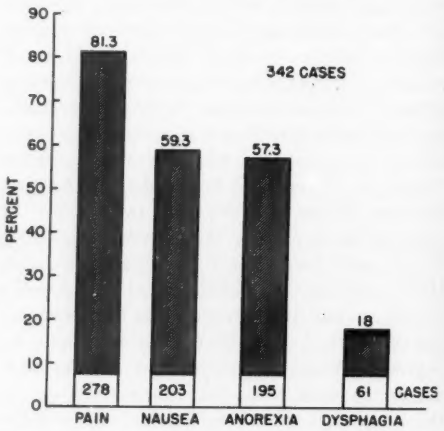

FIG. 7. Distribution of symptoms in 342 cases of carcinoma of the stomach.

of 25 pounds in 7 months. The other signs in order of frequency were tenderness (47 per cent), simple vomiting (45 per cent), palpable mass (41 per cent), retention vomiting (21 per cent), melena (20 per cent) and palpable spread (17 per cent) (fig. 8).

The most frequent pathologic type was diffuse involvement (66 per cent) followed in frequency by ulcerative lesions (23 per cent) and polypoid lesions (11 per cent) (fig. 9). Probably the reason that approximately one-fourth of our patients had ulcerative lesions is that in some of these patients it was impossible to determine preoperatively whether the ulcer was malignant or benign, and only after microscopic examination after gastrec-

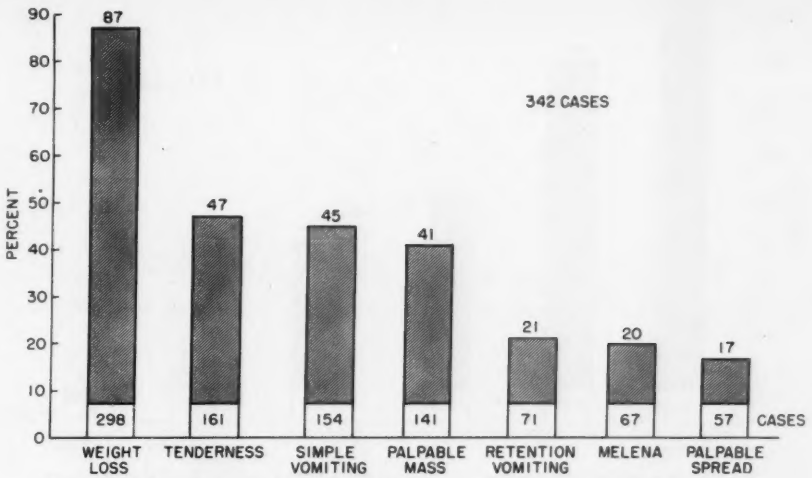

FIG. 8. Distribution of physical signs in 342 cases of carcinoma of the stomach

tomy could the malignant lesion be correctly diagnosed. These cases offer a particularly good prognosis and the more such cases in a series, the better the results will be.

It is the present concensus that the only curative therapy of gastric carcinoma is gastrectomy. Unfortunately, far too frequently the lesion is inoperable when the patient first consults a physician. Of our 342 patients, 42 (12.2 per cent) had inoperable lesions when first seen. Although 88 per cent were considered to have operable lesions, 26 (7.6 per cent) of the total refused surgical treatment and 274 (80 per cent) of the total were operated upon. Less than half of these (138 or 40.4 per cent of the total) were resectable (fig. 10). Of the 138 patients in whom resection was possible, the lesion had extended beyond the stomach (to the regional lymph nodes, adjacent viscera or liver) in 102 (74 per cent). In only 36 (26 per cent) of those resected and 10.5 per cent of the entire group was the lesion apparently limited to the stomach (fig. 11).

Most of the patients in whom resection was possible were treated by radical subtotal resection of the distal portion of the stomach, as previously described by us. Of the 138 patients, 91 (66 per cent) had such resections, 29 (21 per cent) had total gastrectomies and 18 (13 per cent) had proximal subtotal gastroesophagectomies for carcinoma of the gastric cardia (fig. 12).

The operative mortality in the entire resected group was 10.8 per cent. It was lowest in those

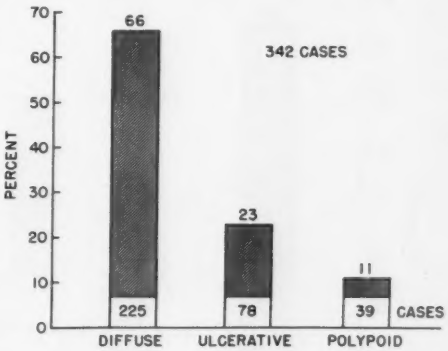

FIG. 9. Distribution of 342 cases of carcinoma of the stomach according to gross pathologic type of lesion.

patients subjected to distal radical subtotal resection (7.7 per cent), next highest for total gastrectomy (10.3 per cent) and highest for proximal subtotal resections (27.7 per cent) (fig. 13).

Of the 342 cases, 264, which represents 98 per cent of those seen more than 5 years ago, have been followed. None of the 162 with nonresectable lesions survived for 5 years. Of the 264, 27 (10 per cent) survived for 5 years. Of the 102 in whom resection was possible, 27 (26 per cent) survived for 5 years. Of 71 who had palliative resections (the lesion had extended beyond the stomach), 9 (13 per cent) survived for 5 years and of 31 whose

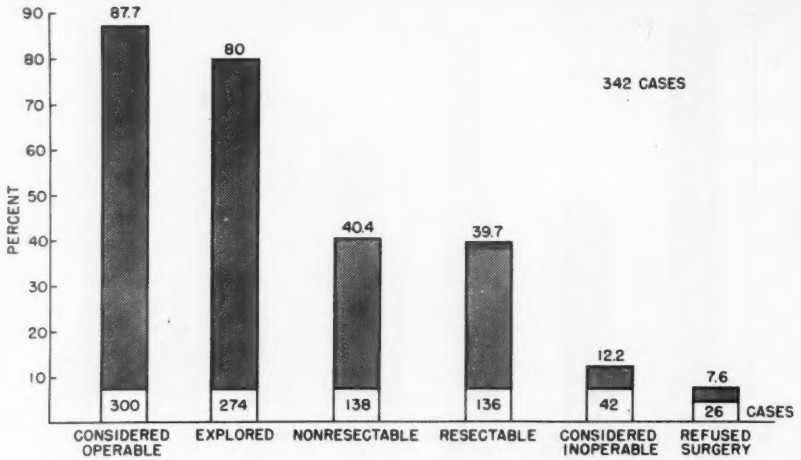

FIG. 10. Method of management in 342 patients with carcinoma of the stomach

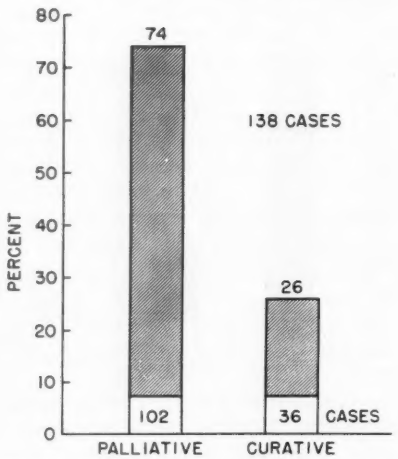

FIG. 11. Classification of resective procedures performed on 138 patients with carcinoma of the stomach.

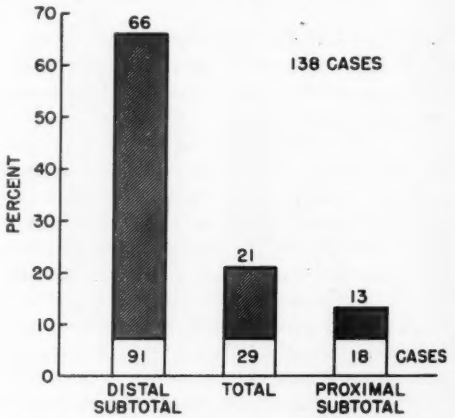

FIG. 12. Type of resection in 138 patients with carcinoma of the stomach.

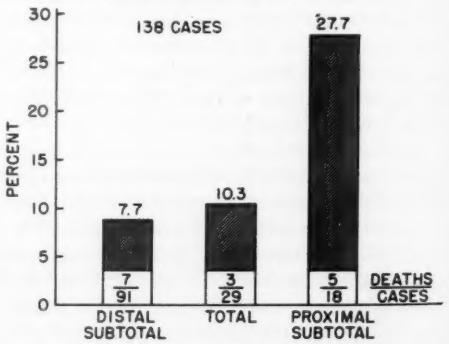

FIG. 13. Operative mortality according to type of resection in 138 patients with carcinoma of the stomach.

lesions were apparently limited to the stomach, 18 (58 per cent) survived for 5 years (fig. 14).

Of the 342 patients 80 per cent had exploratory operations, 40 per cent had resections, 10 per cent were alive after 5 years and 7.5 per cent were alive after 10 years (fig. 15).

The survival rate in gastric cancer falls off rapidly after various types of therapy until approximately the third year, when a plateau is reached, after which there is relatively little further decrease in survival rates (fig. 16). Thus, a patient who is apparently free from disease 3

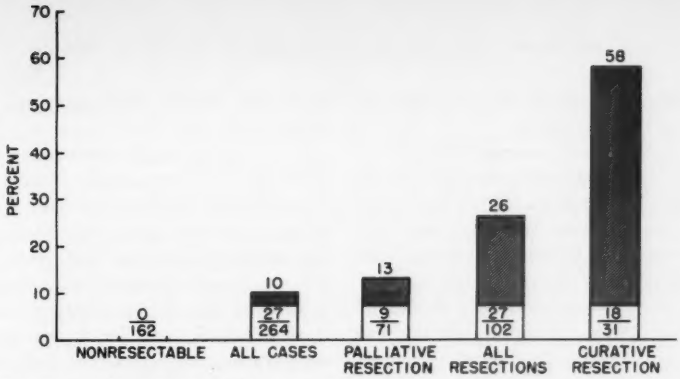

FIG. 14. Results in 264 patients with carcinoma of the stomach who were followed for 5 years

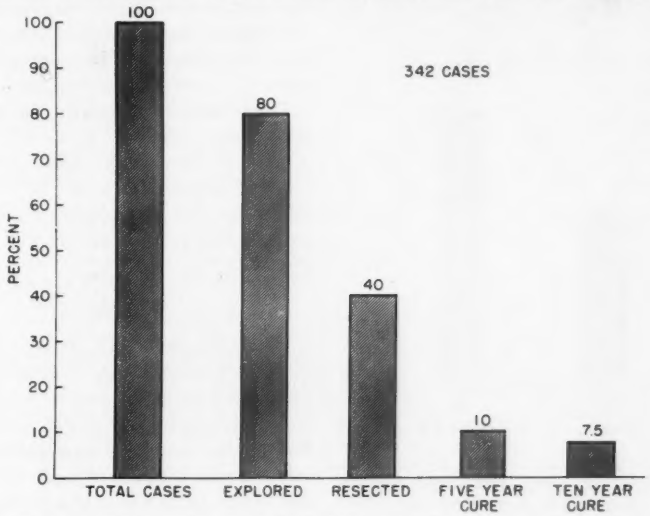

FIG. 15. Operability and end results in 342 cases of carcinoma of the stomach

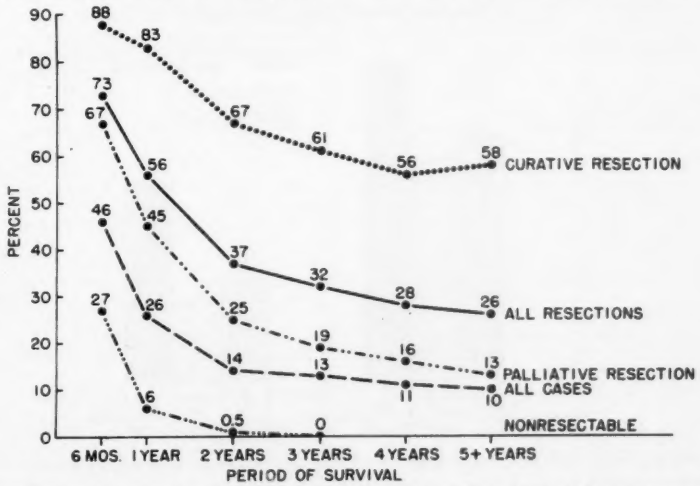

FIG. 16. Survival rates according to method of treatment in 336 patients with carcinoma of the stomach who were followed (98 per cent of series).

years after resection for gastric carcinoma has an excellent chance of being cured.

The prognosis in gastric cancer is influenced by many factors. As mentioned previously, paradoxically a short history is associated with a decreased 5-year survival rate. In our patients with symptoms of less than 9 months' duration, the 5-year survival rate was only 7 per cent, whereas in those with symptoms of more than 9 months' duration, it was 16 per cent (fig. 1). Sex also was of prognostic significance. Of the 181 men seen more than 5 years ago, 16 (8.8 per cent) were alive after 5 years and of the 83 women similarly observed 11 (13 per cent) survived for 5 years (fig. 17).

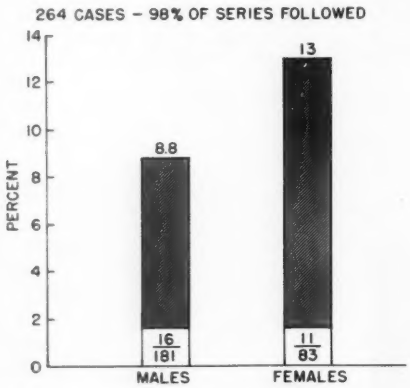

FIG. 17. Five-year survival rate according to sex in 264 patients with carcinoma of the stomach who were followed (98 per cent of series).

The amount of weight lost was also of prognostic significance. Of 264 patients observed for 5 years or more, 171 had lost less than 25 pounds, of whom 12 per cent survived for 5 years; 93 patients had lost more than 25 pounds, and of these, only 6 (6.4 per cent) survived for 5 years. Weight loss apparently was of less prognostic significance in women than in men. The 5-year survival rate in women who had lost less than 25 pounds was 15 per cent, and in those whose loss was more than 25 pounds it was 10 per cent (fig. 18).

Of tremendous prognostic significance was the location of the lesion. None of our patients with lesions in the cardia survived for 5 years.

The type of lesion greatly influenced the prognosis, which was best in ulcerative lesions and poorest in those with diffuse infiltration with polypoid lesions in between (fig. 4). Although age influenced the resectability incidence in our series, it had no effect on the long term survivals (fig. 3).

SUMMARY

The incidence of carcinoma of the stomach is fortunately decreasing, but unfortunately, the prognosis is poor because too frequently the lesion is far advanced when treatment is instituted. A long history is frequently associated with a higher survival rate because, in those with slowly growing lesions or in whom the malignant lesion de-

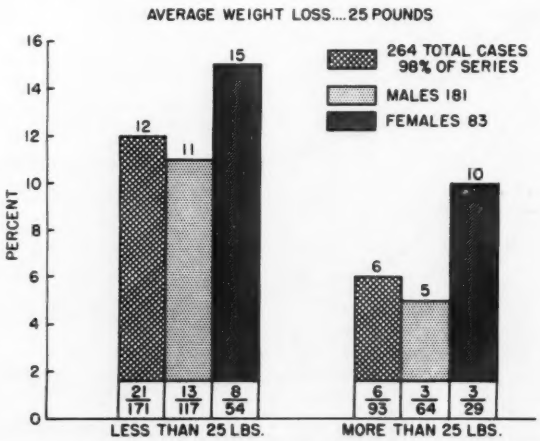

FIG. 18. Five-year survival rate according to amount of weight lost in 264 patients with carcinoma of the stomach.

velops in a pre-existing benign ulcer, the long duration of symptoms is of less importance. However, everything else being equal, the earlier that adequate therapy is instituted, the better the results. In fact, we believe that the abdomen should be explored when a definite diagnosis of cancer cannot be made. This means that exploration is indicated in precancerous lesions, such as polyps, chronic gastritis, especially associated with pernicious anemia, chronic gastric ulcer and even in individuals in the cancer age with persistent gastric symptoms which do not respond to conservative therapy. In our series of 342 patients, 80 per cent had exploratory operations, 40 per cent had resections, 10 per cent survived for 5 years and 7.5 per cent, for 10 years.

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ACUTE CHOLECYSTITIS: OPTIMAL TIME FOR AND EXTENT OF OPERATION*

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The discussion of immediate surgical intervention for acute cholecystitis began with many sources at about the same time.[2-5, 8-10] Chronologically it may be dated from the papers published by Heuer in the early 1930's. Heuer reported the incidence of gangrene and perforation of the gall bladder to be 20 per cent in cases of acute cholecystitis which were treated by the traditional method of waiting for the acute attack to subside, with a mortality of 12.5 per cent to 47 per cent when that complication had occurred. Immediate operation in the acute phase before perforation, on the other hand, was performed with a mortality of 2 per cent. The conclusion was that whenever a patient has acute cholecystitis he should be operated upon promptly. Heuer's reports were substantiated by other authors. They indicated that the patient with acute cholecystitis when treated conservatively was subject to grave complications and considerable hazards. Today discussion continues as to when a patient with an acute attack should be operated upon. When gallstone colic has supervened, everything else being equal and with no contraindications, early operation incurs about the same small risk as does an interval cholecystectomy. Obstruction, edema, secondary infection and other complications increase the risk as they progress. The discussion of this question was soon expanded as it became common knowledge that some gall bladders in the acute phase pose tremendous technical difficulties with edema, induration of tissues and the difficulty of identifying the important duct structures, thus inviting tragic injury of the common duct. Moreover, some of the patients were so ill that insistence on proceeding with cholecystectomy under almost moribund conditions would at times obviously lead to a fatality which could have been avoided in certain instances by a less extensive technical procedure, namely a drainage of the gall bladder and the surrounding inflamed area. We wish to report our experience with and our attitudes toward these problems in judgment and technique.

During the 16-year period from 1944 to 1959, we performed a total of 522 operations for benign lesions of the biliary tract. These included operations for stones in the gall bladder with or without acute inflammation, operations for stones in the common bile duct and the much less common operations for acute inflammation of the gall bladder without stones. Also included were the soft tissue obstructive lesions of a benign character at the lower end of the common duct which are caused by stenosis of the ampulla of Vater or by pancreatitis. There were 135 operations for acute cholecystitis. The diagnosis of "acute" was based on both clinical and pathologic findings. If the attack was severe colic, the patient was operated upon promptly, cholelithiasis was found, and the pathologic report was chronic or subacute, the case was still included in the series on the assumption that such an attack was the initiation of an acute process which might have gone on to empyema, but which was interrupted before it progressed to that stage. Also included are the cases pathologically diagnosed as acute even though the clinical diagnosis was subacute or chronic. These 135 cases represent 26 per cent of the entire series of benign biliary tract lesions in the surgical group.

AGE AND SEX

The youngest patient operated upon for acute cholecystitis was 20 years old and the oldest was 90 years old. The decade with the largest number of patients was that between age 41 and 50 in which there were 44 patients, but it is also significant that the 35 patients over 60 years of age comprised 26 per cent of the group. Fifty-six (41.5 per cent) of the patients were men (table 1). This is a higher relative proportion than is reported in most series.[1]

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TABLE 1

Sex and race distribution of 135 acute cholecystitis patients

	Number	Per Cent
Male	56	41.5
Female	79	58.5
White	133	
Negro	2	

TABLE 2

Biliary tract operations: benign lesions 1944–1959

	Number	Per Cent
Total operations (acute and chronic)	522	
Acute cholecystitis	135	25.8
Cholecystectomy	130	96
Cholecystectomy only	97	
Cholecystectomy and choledochostomy	33	24.4
Cholecystostomy	5	3.7
Deaths	1	0.7

CHOLECYSTECTOMY

In 130 cases of acute cholecystitis, approximately 96 per cent of the group, cholecystectomy was accomplished (table 2). In 33 cases (24.4 per cent) choledochostomy was performed in addition to cholecystectomy; thus in 96 per cent of the cases a definitive operation was performed. In five instances (3.7 per cent) the patient was considered too ill for a more extensive operation and cholecystostomy alone was accomplished. In the entire series there was 1 postoperative death, a mortality rate of 0.74 per cent. This death occurred in a 61-year-old woman who was desperately ill (really moribund) at the time of operation. At operation, perforation of the common duct was found with a retroperitoneal extravasation of bile; choledochostomy and cholecystectomy were performed. The patient died 24 hours later.

CHOLEDOCHOSTOMY

Choledochostomy was performed in 33 patients from the entire group (table 3). This represents an incidence of exploration of the common duct of 24.4 per cent, a slightly lower figure than was found in our general series of 522 operations for benign lesions of the biliary tract in which the incidence of common duct exploration was 28 per cent. But in the group in which the common duct was explored, stones were found in 16, that is, 48.5 per cent of those explored, a higher incidence than the 35 per cent incidence that we found in the general group in which chronic as well as acute cases were included[7]. It is interesting also that since we do not hesitate to open the duodenum when we suspect soft tissue obstruction or retained stones,[6, 7] the combined supra- and transduodenal approach was employed in 10, or 30 per cent, of the common duct explorations, and stones were found in 70 per cent of these; whereas the supraduodenal approach was used in 23 instances, or 70 per cent of the cases, and stones were found in 39 per cent of these. There were no deaths in the group in which the duodenum was opened. Two patients without stones had subsiding acute pancreatitis, and 1 patient without stones had soft tissue obstruction of the distal common duct. We would caution that in certain instances when there is tremendous edema in the gastrohepatic omentum and in the area of the duodenum, it may be inadvisable to open the duodenum; careful judgment must be used as to the indication for this additional approach.[7] It is noteworthy, however, that in the entire series of 522 cases with 136 common duct explorations, we have not left a residual stone in the common bile duct when the combined supra- and transduodenal approach was used, but we have had this unfortunate experience three times when only the supraduodenal approach was used.

TABLE 3

Distribution of stones in 135 acute cholecystitis patients

	Number	Per Cent
Stones in gall bladder	130	96.3
Stones absent	5	
Stones in common duct	16	11
Choledochostomy	33	24.4
Stones (stones 11% of entire series)	16	48.5
Supraduodenal approach	23	70
Stones	9	39
Combined supra- and transduodenal	10	
Stones	7	70

Stones were recovered from the common duct in 11.1 per cent of the patients in the acute

TABLE 4

*Cholecystostomies: 5 acute cases**

	Number
Patients desperately ill	5
Local anesthesia	3
General anesthesia	2
Stones in gall bladder	5
Perforation of gall bladder	2
Results	
Good; no recurrence of symptoms	4
2nd Operation 7 months later for obstructive jaundice; died, cardiac, in 1 week	1

* Ages of patients: 90, 85, 83, 80 and 75 years.

group, whereas in the entire series, the chronic and the acute combined, stones were found on common duct exploration in 9.8 per cent.[7]

CHOLECYSTOSTOMY

The 5 patients who had only cholecystostomy (see table 4) were desperately ill, and the extent of operation was decided upon prior to surgical intervention because it was believed that they could tolerate no more extreme procedure. In 3 patients, local anesthesia alone was used, and in 2, general anesthesia was employed because there was some uncertainty as to the preoperative diagnosis. Of the five patients, four had good results and have not been reoperated upon since, but 1 within 7 months had obstructive jaundice and was again operated upon; cholecystectomy and choledocholithotomy were done. The patient died 1 week later of cardiac failure. He was an 83-year-old man who was known to have cardiac symptoms and to be a poor risk.

A brief resumé of the 5 cases in which it was elected to do cholecystostomy follows. The condition of a 75-year-old woman, gravely ill and disoriented, became worse with conservative treatment, and cholecystolithotomy and cholecystostomy were performed under local anesthesia in 1957. She recovered. She had some symptoms related to postoperative incisional hernia but no real biliary tract symptoms since the operation. Now, 3 years postoperatively, she is confined to a nursing home with cardiac and psychiatric symptoms.

In 1952 a second patient, an 80-year-old woman, had a cholecystostomy. General anesthesia was used because of some uncertainty as to the preoperative diagnosis. She was desperately ill. A cholecystolithotomy and cholecystostomy were done. Now, at age 88, she experiences no biliary tract symptoms of importance, but she is an aged invalid.

A third woman, age 90, was operated upon in 1955 after being desperately ill and becoming worse under conservative measures. Cholecystolithotomy and cholecystostomy were done under local anesthesia. The result was good. She died 3 years later of a heart failure.

A fourth patient, an 85-year-old man, was disoriented and desperately ill. The operation planned was cholecystostomy and it was done under local anesthesia. The gall bladder was found to be perforated. The patient recovered but died 1 year later of myocardial infarction; At autopsy no residual gallstones were found and the common bile duct was normal.

A fifth patient, a man aged 83, gravely ill, but with an uncertain diagnosis and consideration of possible appendicitis with perforation, was operated upon under general anesthesia. At operation the gall bladder was found to be perforated. A cholecystolithotomy and cholecystostomy were done. He recovered from this acute attack but 7 months later because of obstructive jaundice he was operated upon again. A cholecystectomy and choledocholithotomy were performed. He died 1 week later of cardiac failure.

Thus, of the 5 patients who underwent cholecystostomy because it was judged that they were too sick to tolerate anything but a short drainage procedure, all recovered following this operation. But, as will be seen, 1 of them was not cured. Stones remaining in the common bile duct necessitated a secondary operation, and from this operation the patient died. Thus in the sequence and follow-up it becomes apparent that the mortality rate of managing cases in this manner was at least 20 per cent. Had these patients all been operated upon more drastically and cholecystectomy and, in the instance of common duct stones, choledocholithotomy performed, the mortality rate may well have been as high as 50 per cent. It is only fair to add the two operations together in computing the mortality rate for this type of management when there is 1 incomplete procedure and the necessity of a subsequent definitive one, instead of 1 definitive operation the first time.

TABLE 5

Acalculous cholecystitis: 5 acute cases

Disease	Procedure	Sex	Age
			years
Acute cholecystitis and acute pancreatitis	Cholecystectomy and transduodenal ampullotomy	M	65
(2) Gangrenous cholecystitis; no stones	No common duct exploration	M(2)	42 and 38
Acute cholecystitis, with stenosis	Cholecystectomy, combined supra- and transduodenal exploration of common duct, and sphincterotomy	F	27
Acute cholecystitis; common duct normal	Cholecystectomy	F	25

CHOLECYSTITIS WITH LITHIASIS AND ACALCULOUS CHOLECYSTITIS

As is well known, most patients who are operated upon in a stage of acute cholecystitis have stones in the gall bladder, and in reality the disease is one of obstruction of the gall bladder with a superimposed acute inflammatory reaction. In our series, 130 of the 135 patients had stones in the gall bladder. In the remaining 5 instances (3.7 per cent) there was an acute cholecystitis but no stones were found in either the gall bladder or in the common duct (see table 5). This too may be an obstructive phenomena with secondary inflammation or it may be a primary inflammatory reaction. Of the 5 patients, 3 were men (ages 65, 42 and 38). One had acute pancreatitis and an acutely inflamed gall bladder and 2 had gangrenous cholecystitis. The one with pancreatitis underwent exploration of the common bile duct, but exploration of the common duct was not indicated in the 2 with gangrenous cholecystitis. Of the patients without stones, 2 were women. One, age 27, had an acutely inflamed gall bladder without stones in it. The common duct was explored and a transduodenal sphincterotomy was performed for stenosis of the papilla of Vater. The other woman, age 25, had no stones. The common bile duct was not explored.

SEQUELAE

There were no bile duct injuries in this group and there was 1 postoperative death, recorded above. But on follow-up it was ascertained that 6 of the patients required secondary operations. One of these was for incision and drainage of a subphrenic abscess 1 week after operation. Another was a very rare bile peritonitis which developed after a T-tube was removed in the 6 weeks after the operation. The patient had so far made an uneventful recovery. The afternoon that the T-tube was removed he became violently ill with signs of severe peritonitis. Operation was clearly indicated. Instead of having been walled off, the peritoneal cavity proved to be in free communication with the opening into the bile duct and an extensive intraperitoneal bile leak had occurred. Peritonitis had resulted. Drainage resulted in prompt recovery. This is the only case of its sort that we have encountered. The third patient, a 44-year-old woman, had a retained common duct stone after cholecystectomy and supraduodenal exploration of the common duct. Three months later, by a supra- and transduodenal exploration of the common duct, the retained stone was removed. The patient recovered and has been well now for over 2 years. A 22-year-old woman, the fourth patient, had cholecystectomy for acute cholecystitis and cholelithiasis and, because of a persistence of symptoms, underwent exploration 2 years later. Common duct exploration gave negative results. The patient is among those with a poor outcome. The fifth patient requiring secondary operation was a woman who had a cholecystectomy for acute cholecystitis and cholelithiasis, and because of recurrent symptoms a sphincterotomy was done for pancreatitis in May 1953. This patient had a good result following the second operation. The sixth case was that of an 83-year-old man, a cardiac patient, desperately ill, who had cholecystostomy and choledocholithotomy. The

TABLE 6

Follow-up on 135 acute cholecystitis patients

	Number	Per Cent
Followed for at least 1 year	105	
Good results from operation	104	97
Poor results including 2 deaths (one immediate and one 7 months postoperatively)	3	3
Untraced	22	
Died of unrelated cause 2 months postoperatively	1	
Operated upon less than 1 year ago	5	

result was poor. He had a subsequent cholecystectomy and choledocholithotomy seven months later because of obstructive jaundice. He died of cardiac decompensation 1 week after this operation.

FOLLOW-UP

An attempt was made to follow all the patients in December 1959, and a follow-up was accomplished for a significant percentage (table 6). Five operations were performed after March 1959, and therefore, a year had not elapsed since the procedure. One patient died postoperatively, 1 died 7 months later of a secondary operation, 1 patient died of an unrelated cause 8 weeks postoperatively and after leaving the hospital, and 22 were not traced or were lost to follow-up. For at least a year 105 patients were followed. Some of them underwent secondary operations as cited above. The death occurring after the first operation and the death following the secondary operation necessary for obstructive jaundice 7 months after the first operation should be included among the poor results. There were 2 patients who had a variety of symptoms judged to be psychosomatic who have not been classed as bad results from the operations although some of the symptoms referred to the upper abdomen. There is one fat man who continues to have symptoms related to the upper abdomen which resemble the post-cholecystectomy syndrome. Thus only 3 patients (2.8 per cent) of 107, including 2 deaths, are classed as poor results after at least 1 year of follow-up. Ninety-seven per cent of the patients ultimately had good results from the operation.

DISCUSSION

All the cases in this group had absolute indication for surgery. The patients were sick people and the problem was not one of trying to compare the surgical method with a justifiable alternate. Even the most reluctant internist could not present results in 135 comparable cases such as these frequently reported by surgeons. Nonetheless, there is still need for sound judgment in these problems and also for something which is infrequently acknowledged in a paper—skillful technical performance. Both of them must come into play.

We believe that acute cholecystitis is a surgical disease. There may be circumstances which make it wise for the surgeon to defer action, to withhold action or to take limited action. Under certain circumstances it may be evident that the patient at the time that the surgeon sees him is moribund and that apparently no operation can save him. On the other hand, we hesitate to withhold action in even the most desperate cases because we have seen patients who are apparently moribund experience the most remarkable recovery following surgical intervention. Even with high fever, with disorientation, with distention and extreme rigidity, a drainage operation may prove effective for the patient, and therefore, to withhold the procedure simply because one thinks that death is inevitable seems most unwise. The single death occurring in this group was of such a patient, who had an unusual complication of a ruptured common bile duct and extensive extravasation of bile in the retroperitoneal area.

In the treatment of cholecystitis we believe in attempting to carry out a definitive procedure. This means that at times the patient's condition must carefully be worked into the optimal phase. What is the best time for operation? Is it immediate? Is it delayed? This question cannot be answered in either of those terms. It can be answered: When the patient can be gotten in the best position for surgery. This may mean that an operation should be performed after a matter of hours, hours needed for decompression and replacement of electrolytes and fluids. It may mean after watching carefully for several days a very sick patient, as the hope of improvement decreases, and then acting immediately when this hope is lost. Thus it may be hours or days before the operation should be undertaken.

Certainly it is no longer reasonable to insist that a patient should not be operated upon during the first week of the disease if he is not operated upon in the first 24 to 36 hours. Many such patients have had the acute phase of the disease for several days and the operation can be undertaken within a few to 24 hours as an optimal time for its performance.

There were relatively few cholecystostomies in this group, less than 4 per cent. In the literature, the incidence of this procedure in acute cholecystitis is two or three times greater, and in some instances, it has been reported to be as high as 25 per cent of the cases. We do not subscribe to this. We would prefer complete action if it can be done, leaving the patient with a reasonable chance of recovery. If it is not done, the risk of a secondary operation is added, for it is well known that in a high percentage of those cases in which cholecystostomy is done, secondary operations are not only advisable but are mandatory.

As to the technical aspects, this can be a most difficult operation. There may be a great deal of edema and induration, making exposure of the common bile duct most difficult. We suspect, and our suspicions are well founded, that in some residency training programs the incidence of injury to the common bile duct in operations for acute cholecystitis may be as high as one in 100. At the residency training level, when the men are developing technical skills, it should be emphasized that unless one clearly sees the common bile duct, not only the common bile duct, but the common bile duct above and below the cystic duct, then a cholecystectomy should not be performed. In other words, if the structures cannot be identified, then one should make a delaying action and perform a cholecystostomy rather than run the dreadful risk of injuring the common bile duct. The indications for action, therefore, are not only determined by the condition of the patient, but are determined by the skill of the surgeon as well, and they may differ in the same surgeon at different periods of his career. As he grows in experience and ability he may be able to accomplish what he could not have done years earlier. But the welfare of the patient demands that the surgeon act not solely by a rule, but by wise judgment, considering all factors. Thus an operation which was not originally "planned" as a cholecystostomy for acute cholecystitis may nonetheless be converted into just that degree of surgical procedure and no more. In this group of cases we were fortunate in not having to deviate from the originally planned procedure, and thus we performed cholecystectomies when we had planned to do them, and we performed a less definitive operation only in the cases in which the patient prior to the surgery was judged to be too ill to tolerate a more extensive procedure. Among those in whom we have done cholecystectomies were even patients with gangrene of the gall bladder. Thus it is possible and justifiable to perform cholecystectomies or definitive operations even on patients with gangrenous gallbladders, but in this instance, as with all these procedures, there may be great distortion of tissues from edema, considerable induration and much difficulty in identifying structures, and we would warn again that unless the structures can be identified cholecystectomy should not be performed. The technical device of removing the gall bladder from the fundus down to the ampulla, to be sure, was employed in a considerable number of these patients, but even with this procedure, we feel that it is necessary for the surgeon to identify the common bile duct and clearly to know the position in which he is working in at all times with respect to the duct.

SUMMARY

In 522 operations for benign lesions of the biliary tract, acute cholecystitis was encountered in 135 cases (25.8 per cent). Cholecystectomy was performed in 130 patients (96 per cent) and the common duct was explored in 33 (24.4 per cent). Cholecystostomy was performed on 5 desperately ill patients, all of whom recovered. There was 1 death in the entire group, a mortality rate of 0.74 per cent. Good results from the operation were obtained in 97 per cent of the patients.

We believe that cholecystostomy for acute cholecystitis should be reserved for the desperately ill patient and that most patients can be prepared for definitive operation (cholecystectomy). We regard all these patients as surgical cases, and feel that the decision as to when the operation is to be done, whether within hours or days after admission of the patient to the hospital, is one of circumspection. Operation should be performed only after measures to improve the patient and get him into optimal position have been put into effect.

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SURGICAL COMPLICATIONS OF STAPHYLOCOCCIC PNEUMONIA IN INFANTS AND CHILDREN*

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Staphylococcic pneumonia and its complications constitute the most severe and probably the most common serious infection seen by the surgeon in the neonatal and early infancy period. The antibiotic resistance of certain strains of the staphylococcus has contributed to the increased incidence of staphylococcic infections of all types in infancy and childhood. Although the prognosis of pneumonia due to this organism has improved in the past 2 decades, too many of the deaths still occurring in afflicted infants seem to involve the management of pleural complications rather than the pulmonary infection as such. Most of the published articles emphasizing this fact have come from the eastern United States with a few from the Middle West,[9, 17, 20, 33] but recently an increase in reported western cases has been evident.[24] Experience with the surgical care of almost 40 cases during the past 5 years in three institutions in widely separated areas (Children's Hospital, Boston, Massachusetts; Kings County Hospital, Brooklyn, New York; and Children's Memorial Hospital, Oklahoma City, Oklahoma) indicates that primary staphylococcic pneumonia presents a fairly consistent diagnostic and therapeutic problem with modifications by the variable factors of new drugs and developing resistance.

It is the purpose of this paper to review briefly my experience with the surgical problems presented by this disease and to emphasize the practical details of treatment. The major surgical complications have been empyema, pneumothorax and pneumatocele, with fibrothorax, pulmonary abscess and purulent pericarditis less commonly encountered.

CLINICAL FEATURES OF STAPHYLOCOCCIC PNEUMONIA

The common clinical features of staphylococcic pneumonia have changed little in the past 25 years, and our own series fits well into the picture described by several authors.[4, 17, 22, 32, 38] More than three-fourths of our cases occurred in infants less than 1 year old. Most were bronchogenic in type, and a moderate winter and early spring incidence peak has been evident in all three locales. A predisposing cause or route of entry was evident in about half the cases (*e.g.*, infant pyoderma, maternal and infant mastitis, an institution where such infections were prevalent). Of 22 patients with empyema reviewed in 1957 at Kings County Hospital,[44] 8 developed the infection in one foundling home and 5 while at Kings County Hospital for another complaint. (Perhaps this should be called "staff empyema.") The cases considered were not those of chronically debilitated patients and had no evident relation to known viral infections. Phagic typing has not been available, nor do we have conclusions concerning the significance of the coagulase reaction of the incriminated hemolytic *Staphylococcus aureus*. Several of the patients were already receiving prophylactic antimicrobial drugs, often in inadequate dosage, when their pneumonia became clinically evident.

A very few of our cases were sudden in onset or associated with overwhelming toxicity. In most instances, the first symptoms were suggestive of a mild gastroenteritis or upper respiratory infection and were followed in a few days by development of fever, cough and an increased respiratory rate. Several patients then received an antibiotic with subsequent failure to manifest the more classic signs and symptoms of pneumonia as the diffuse part of the infectious process was controlled. These were often the cases in which complications were first diagnosed by roentgenographic examination. The usual untreated course led progressively to a moderately high fever, cough, dyspnea with rapid grunting respirations and, not infrequently, cyanosis. Anorexia, diarrhea, abdominal distention, lethargy and irritability were fairly common; convulsions occurred in a few. The physical findings were usually those of a unilateral consoli-

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dating process, but only prompt and serial use of chest x-rays allowed a reasonable picture of the intrathoracic progression of disease. Although the respiratory signs were occasionally overshadowed by the toxemia, most of the severe cases presented a fairly typical appearance at surgical consultation: the infant was pale, somewhat cyanotic, with distressing cough and grunting labored respirations, and often with moderate to marked abdominal distention. An enlarging liver, tachycardia and moist rales suggested congestive heart failure in several infants with extensive pneumonitis. Labored respirations were on a few occasions associated with sternal retraction, but no major obstructed airways were demonstrated in any case. The development of an empyema made the infant seriously ill with tachypnea and a "septic fever." Frequently the situation was suddenly complicated by the formation of a bronchopleural fistula and development of pyopneumothorax. A tension pneumothorax could quickly ensue with mediastinal shift, impaired venous return and other sequelae. It deserves major emphasis that this sequence of events often occurred with startling rapidity even in the known sick infant under care, and in several with a less acute clinical illness, a tension pneumothorax apparently developed from sudden rupture of an air-containing cystic lesion into a pleural cavity as yet manifesting little more than a localized pleuritis.

Leukocytosis was often in the 20,000 to 25,000 range, but a few infants presented a low or normal count, always associated with fairly massive empyema but not necessarily with an unfavorable prognosis. A few limited special studies and the prompt rise in leukocyte count after drainage suggest marrow depression rather than sequestration of blood constituents as an explanation, but more extensive investigation of this picture in youngsters with massive collections of pus is needed.

Anemia was not infrequent on admission of patients and was almost always present (often in the 7- to 8-gm. range) by the time of surgical consultation because of continuing infection, with limited nutritional intake and the unreplaced loss of blood incident to laboratory sampling. Bacteriologic studies have provided puzzles as well as answers. Hemolytic *S. aureus*, coagulase-positive, was usually obtained from the nose and throat, but in the absence of phagic typing this finding is of doubtful significance.[3, 19, 33] Blood cultures were occasionally positive. Smear examination of the empyema on occasion revealed gram-positive cocci even when no organism could be grown by culture. Examples were found in which both sterile and positive loculations existed in the same hemithorax. The lung puncture technique was not used.[13] Only about three-fifths of the staphylococcic pneumonias encountered on the pediatric services provided specific bacteriologic proof of hemolytic *S. aureus*, but the typical clinical and especially roentgenographic features completed the diagnostic criteria. Sensitivity tests were done in nearly all cases and showed a fairly high incidence (50 to 75 per cent) of penicillin resistance. Only 4 of the 13 previously described institution-acquired cases in Brooklyn were penicillin sensitive, although 6 of the 9 home-grown organisms were sensitive. Erythromycin and chloramphenicol were most consistently effective *in vitro*, with bacitracin disappointing in about half of those tested.

PATHOLOGY AND PATHOGENESIS

The characteristic pathologic pattern found in patients dying of staphylococcic pneumonia offers a reasonable explanation of the roentgenographic features and the pattern and frequency of surgical complications.[4, 22, 32] Basically staphylococcic pneumonia infections show two general patterns. The first is the occasionally acute fulminating disease when overwhelming toxicity and suffocation from widespread hemorrhagic and emphysematous pulmonary changes lead to death before tissue necrosis can take place. The second is a suppurative process, usually showing diffuse or localized involvement of pleura. This is the pattern which usually leads to surgical complications and occurs chiefly by bronchogenic invasion with infection descending from the upper respiratory passages into the smaller passages which the organisms usually invade, forming small abscesses in the distal bronchial walls (fig. 1*a*, *outer zone*). Perhaps the striking predilection of the disease for the small infant is as much due to his inherently small air passages as to his immunologic deficiencies. The necrotizing foci set up in the walls of these smaller bronchi can lead to extension to the periphery by way of lymphatics, in which case only pleuritis or empyema may result. This tendency to involve bronchopulmonary segments can give rise to rather

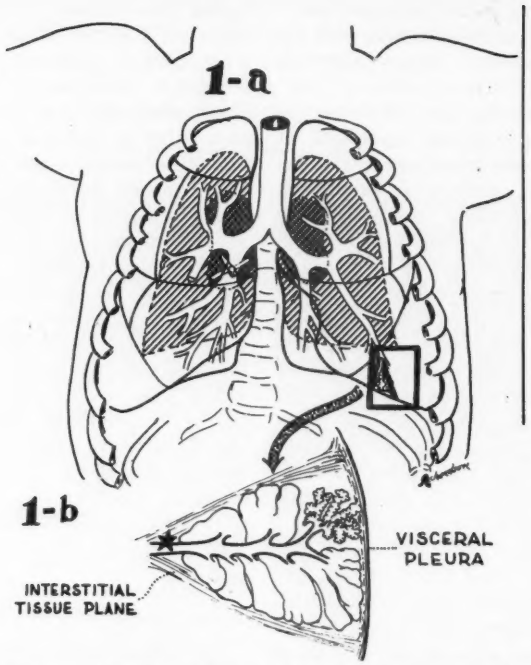


FIG. 1. Diagrammatic presentation of some anatomical factors of importance in shaping the course of staphylococcic pneumonia and its complications. (*a*) Indicates the hilar, central and peripheral or subpleural zones of the lung fields, especially as correlated radiologically. The latter is most commonly involved by the bronchogenous invasion of the primary suppurative pneumonias. A pulmonary lobule is enlarged in (*b*) to indicate a route that could explain the sudden rapid development of huge pneumatoceles or a tension pyopneumothorax. A necrotizing invasion of the small bronchial wall at the apex of the cone-shaped architectural segment (★) could easily dissect and distend the low resistance interstitial tissue plane and also explain the remarkable infrequency of significant lasting pulmonary sequelae.

characteristic cone-shaped areas of parenchymal infiltration (fig. 1*a*) with the large pleural surface thus contacted possibly explaining part of the high incidence of empyema. In other cases, the bronchial wall foci may penetrate or perforate into the interstitial tissue with the formation of multiple tiny abscesses around smaller bronchi in the peripheral lung. As these enlarge, they coalesce to form sizeable cavities, and being near the surface of the lung, the larger abscesses tend to rupture into the pleural cavity, discharging several ounces of pus and usually at least a small amount of air. A bronchopleural fistula with pneumothorax results when the ruptured abscess connects with a bronchus and a tension pneumothorax ensues fairly frequently because of the valvar potential of the edematous bronchial wall with its necrotic perforation. In a number of cases, especially in infants in whom a less peripheral bronchus may be involved, the bronchial wall focus may rupture into the interstitial tissue near the apex of a cone-shaped pulmonary lobule (fig. 1*b* ★). Trapped inspired air, aided perhaps by the patient's coughing pressures, can dissect into the interstitial plane with the dramatic, dangerously rapid development of a huge tension bleb, or pneumatocele, which may or may not promptly perforate into the pleural cavity. It seems unlikely that bronchial intraluminal valvar obstruction with distal distention even with associated necrotizing infection could explain the rapidity of this particular development. The mechanism of bronchiolar and small bronchial dilation beyond check-valvar intraluminal obstruction described by Conway[12] and Caffey[8] (fig. 2) is also probably active in the production of some of the smaller and especially earlier and multiple cystlike hyperventilated

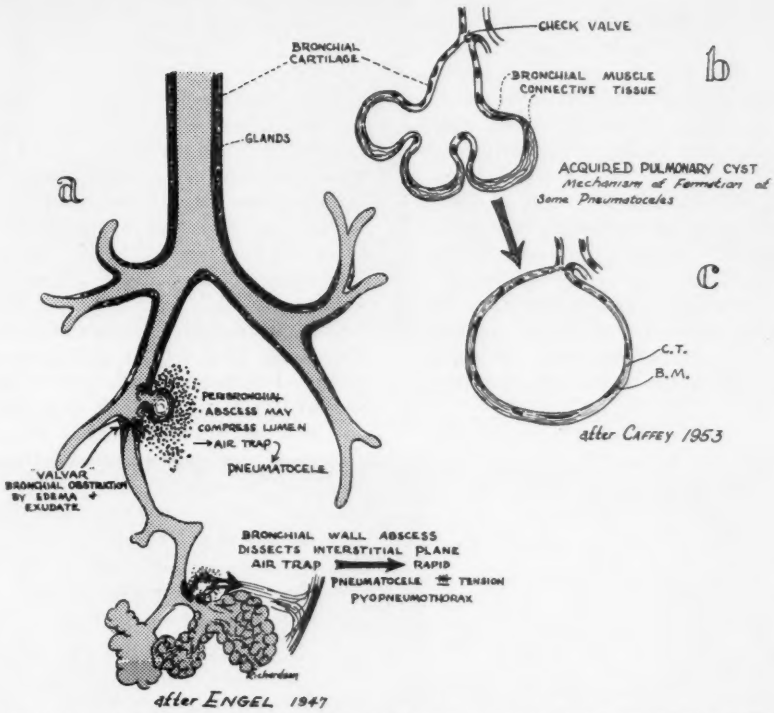


FIG. 2. Pathogenetic mechanisms involved in producing some of the acute complications of staphylococcic pneumonia. A schematic presentation emphasizing the check-valve mechanism active when both the interstitial tissue and bronchioloalveolar sites are considered. In order to have (*a*) correspond to the Caffey[8] schemata (*b*) and (*c*), the intraluminal obstruction site had to be sufficiently proximal to find bronchial cartilage. Caffey illustrated an earlier stage with early alveolar dilation but bronchi unaffected, but Conway[12] found the reverse in some of his cystlike lesions. Stages (*b*) and (*c*) would seem rare in most cases of acute staphylococcic pneumonia.

rarefactions. Enlarging peribronchial interstitial abscesses may even obstruct or involve directly the bronchioles or small bronchi, thus contributing indirectly to the same bronchoalveolar distention.

By contrast, the pulmonary lesion of hematogenous origin rarely leads to complications such as empyema and bronchopleural fistula or even pneumatocele. The often inadequate protection of antimicrobials early in the course of the pneumonia may prevent a fulminating course but probably allows small abscesses to smolder in the lung parenchyma. Persistence with enlargement of these abscesses is aided by the characteristic localization of the Staphylococcus in a relatively avascular focus with consequent inaccessibility as an explanation for the frequent failure of drugs of high activity *in vitro* to eradicate the infection. The intense necrosis with abscess formation can on rare occasions involve an entire lobe or even lung.[31] Sabiston[40] described a small number of cases in which "a pneumatocele became filled with purulent material and behaved as a lung abscess," and he described a case that permitted successful drainage with a trocar and intercostal catheter. It seems preferable to think of such a lesion as a pyopneumatocele rather than as a lung abscess because of both the pathologic connotation and therapeutic implication.

RADIOLOGIC FINDINGS

Correlation of the radiologic and pathologic features of staphylococcic pneumonia in infancy permits a clearer, dynamic concept of the pathogenesis of the clinical events and variations encountered. There are several distinctive x-ray

findings in this disease that are found in bacteriologically proved cases and rarely found with pneumonias of other etiology (table 1). Initially, the chest x-ray may either be normal or reveal only a diffuse haziness or a "faint focal mottling of the lung parenchyma."[19] This may progress rapidly so that in a very few hours there are scattered areas of infiltration, without the peribronchial pattern of aspiration pneumonia, and soon thereafter possibly a rapid accumulation of pleural fluid that may completely obscure developing parenchymal changes. The most distinctive feature of this over-all picture is the rapid changing of the pattern with variations in sequence and completeness, with variable and even paradoxical correlation with the clinical course, and with multiple stages demonstrated simultaneously in the same lung field. The rarity of the occurrence of bilateral parenchymal or pleural disease is fortunate and unexplained. Campbell[9] has pointed out that the distribution of the infiltrative parenchymal changes seen on roentgenograms indicates the presence of multiple inflammatory occlusions in the bronchi of the second and third order.

TABLE 1

Roentgenographic features of acute staphylococcic pneumonia and its complications

1. Rapid change
2. Pneumatoceles (including "lobular ectasia")
3. Infiltration: focal, segmental, lobar, mixed types (variable, bizarre)
4. Pneumothorax—bronchopleural fistula, tension
5. Pleuritis and empyema—diffuse, localized; pneumothorax
6. Emphysema, alveolar; segmental or diffuse, interstitial

At a slightly later pathologic stage, coincident with bronchial and some alveolar necrosis plus developing obstructive elements, Neuhauser and Wittenborg have labeled a roentgenographic picture of "lobular ectasia."[19] This should be considered a stage of multiple small bleb formation occurring relatively early in the course of disease and most often apparent in the few days after the re-expansion of the lung subsequent to adequate drainage of the empyema which may either have obscured or prevented by compression the development of the lesion (fig. 4*f*). Here again two sites for the air valve mechanism are probable—bronchial wall perforation and lumen. Such rarefied emphysematous tension areas cannot adequately use the collateral ventilatory route for decompression because most of the adjacent lung tissue is consolidated. Published

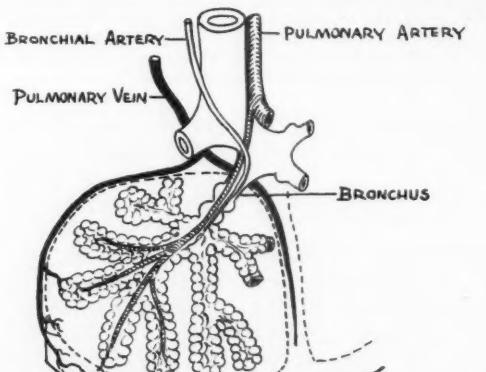


FIG. 3. A diagrammatic illustration of the intimate anatomical relationship of the bronchus or bronchiole and blood supply (after Betts and Bremer[2]) to a pulmonary lobule. Betts has explained the almost constant peripheral subpleural location of the classic pulmonary abscess (anerobic, postaspiration) by postulating this sequence: septic bronchial obstruction → infective thrombosis of the lobular blood supply → dissolution and abscess first manifest in the region of poorest blood supply (subpleural). The usual pyopneumatocele that may be confused with such a lung abscess is very rarely associated with the parenchymal damage, early or late, that seems to result from such an infarction mechanism.

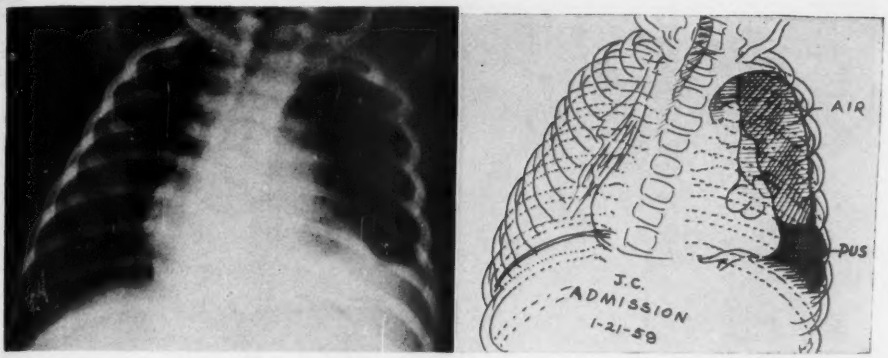


FIG. 4. Roentgenograms of J. C., an 18-day-old infant with a 2-day history of cough and a sibling with pustular skin lesions which cultured hemolytic *Staphylococcus aureus*, coagulase-negative, as did the patient's pleural exudate. *A*. Chest films on admission revealed pyopneumothorax with minimal mediastinal shift but severe respiratory distress. Right lung appears uninvolved.

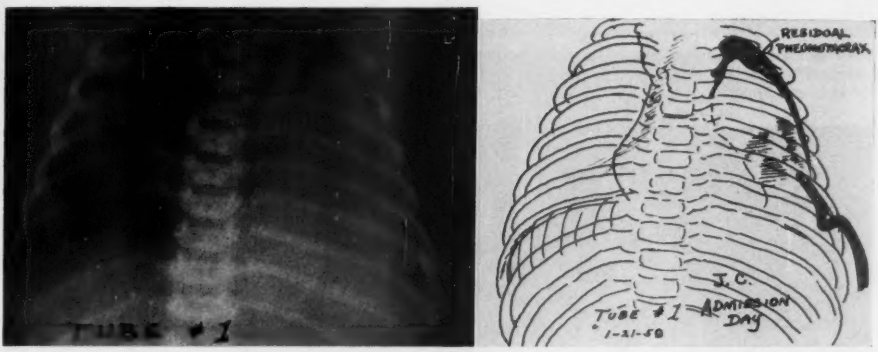


FIG. 4*B*. Immediate post-thoracostomy chest film re-expansion correlated well with the dramatic clinical improvement associated with the evacuation of a moderate amount of air and pus. Tube was only 10 French, and the seventh interspace could prove to be too low to continue effective drainage once the diaphragm rises following evacuation. Irrigation and positioning here might have completed the expansion but the fear of a bronchopleural fistula flooding and cough plus the exhaustion of the infant deterred us. The infiltrative and emphysematous lung changes are now visible.

reports vary from 17 to 84 per cent incidence of such cystic lesions. The frequency of serial chest x-rays and the promptness of empyema drainage probably act as major determinants in the success in visualizing these early multiple pneumatoceles.

The very feature that renders the earliest pneumatocele potentially dangerous as a precursor of a tension pyopneumothorax will allow minimal pulmonary parenchymal damage to result after even a huge pneumatocele with pleural rupture complications. The interstitial site of the rupture of the bronchial wall abscess enables a ball-valve, air-trap mechanism to rapidly dissect out the perilobular or segmental planes to form a huge emphysematous lesion or, by rupture, a tension pyopneumothorax. Yet collapse and healing of this same interstitial space can leave negligible parenchymal change if pulmonary re-expansion is achieved without undue delay. No long term residuals have been noted in our cases as judged clinically and by standard chest x-rays. Late follow-ups on some of the cases with bronchograms should be of interest and may parallel the few previously recorded which found moderate bronchiectasis in the appropriate areas in some cases.[6, 12] Our experience extends that of Hendren and Haggerty,[19] who found pneumatoceles much less common in older children, and it again emphasizes the role of bronchial obstruction with its exudate and calibre factors. Striking

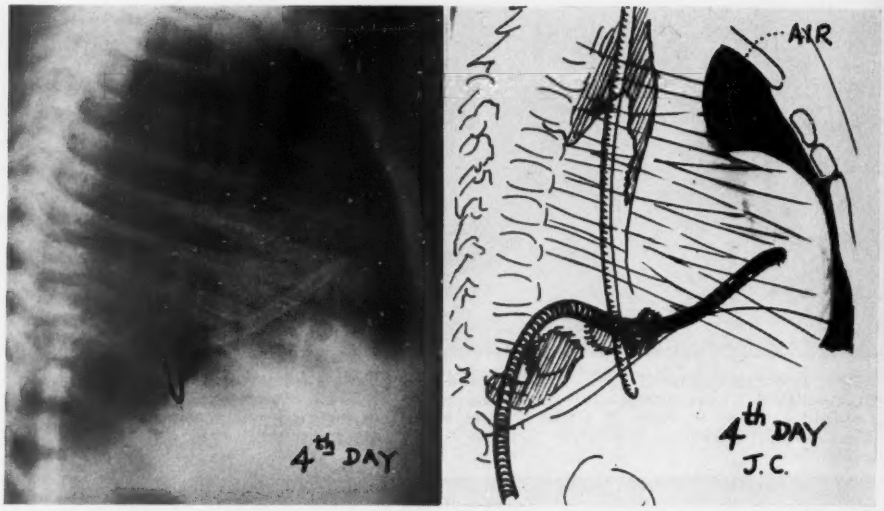


FIG. 4C. Lateral chest film 4 days later demonstrates increased pneumothorax with decreased expansion. Decreased suction air leak suggested tube dysfunction. Nasogastric tube (insufficiently inserted) is evidence that abdominal distension was becoming a factor.

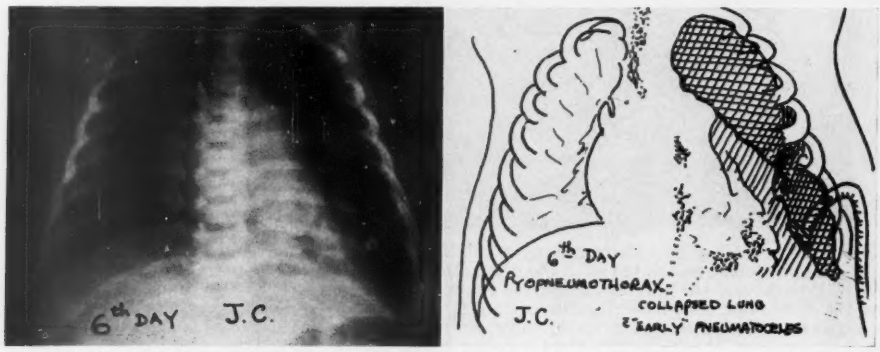


FIG. 4D. On sixth day recurrent pyopneumothorax with collapsed lung had increased. "Lobular ectasia" evident even with the limited expansion. Second tube was overdue.

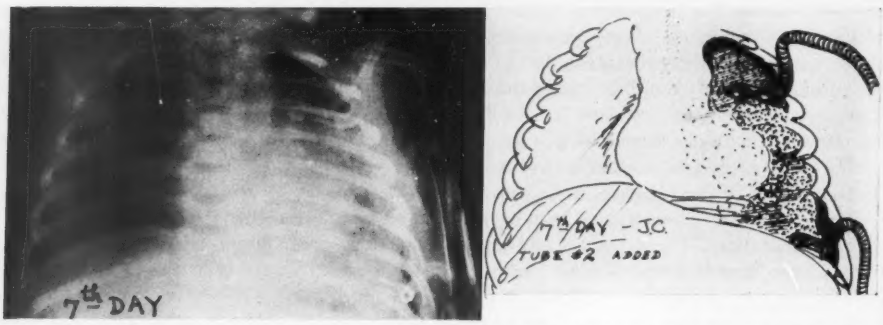


FIG. 4E. By the seventh day a second tube had been inserted via the anterior second interspace and pulmonary expansion was much improved.

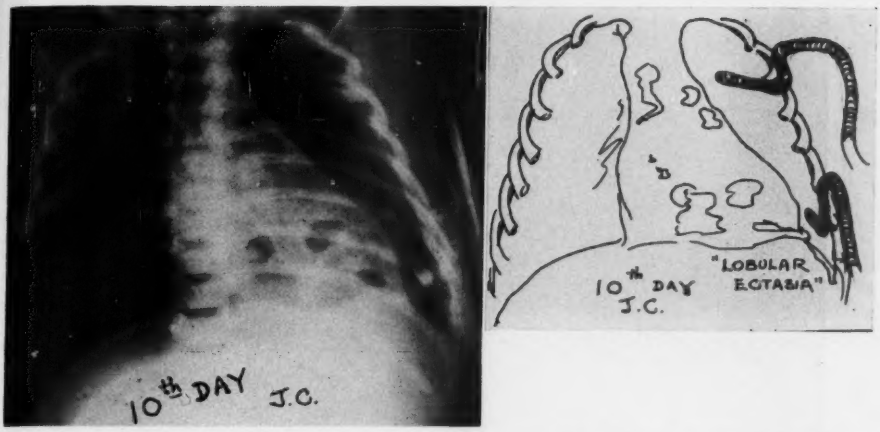


FIG. 4*F*. Chest film on tenth day showed a recurrent pneumothorax due to transient chest catheter obstruction, an indication of the size of the persistent air leak. The "lobular ectasia" is now well demonstrated. Tube manipulation and irrigation → prompt re-expansion.

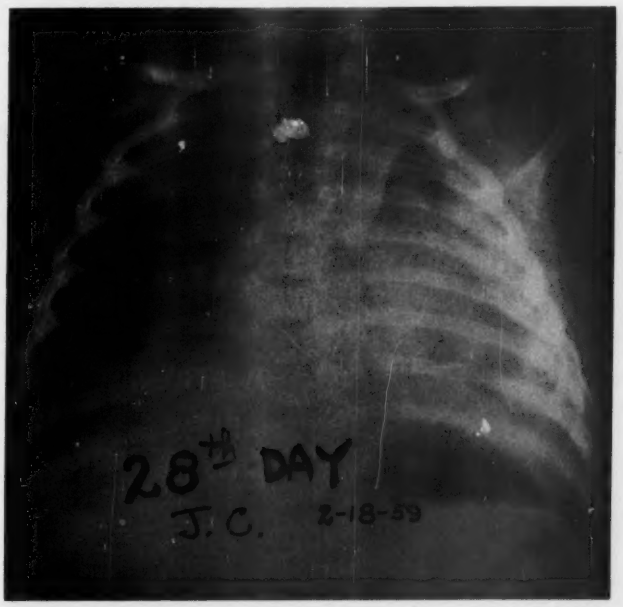


FIG. 4*G*. Both tubes had been out for 1 week at the time of this chest film on the 28th day. Continued clearing of the lung fields with increased expansion is evident along with less mediastinal distortion.

pneumatoceles may persist for weeks in an infant who is afebrile and appears to be well (fig. 5). Roentgenographic signs may take several weeks to subside, but even the residual pleural thickening will disappear in 4 to 6 months, leaving no permanent change in lung or pleura (figs. 4*h* and 6*g*). Spontaneous disappearance of pneumatoceles is usual, but the time of the necessary subsidence of the valve mechanism is not predictable.

Radiographic technical points stressed are the importance of taking upright and *expiration* films to aid in demonstrating small degrees of

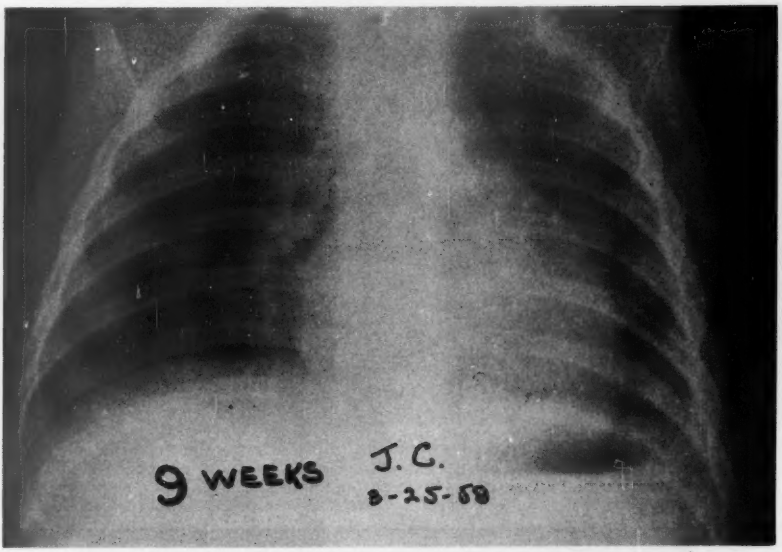


FIG. 4*H*. At 9 weeks there remains only slight apical haziness of significance, and 3 months later no abnormalities were noted.

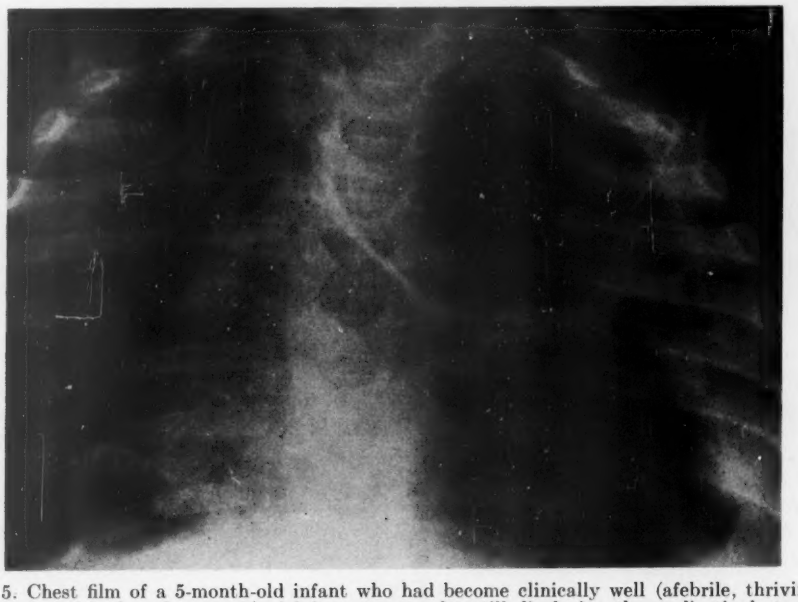


FIG. 5. Chest film of a 5-month-old infant who had become clinically well (afebrile, thriving, no leukocytosis) with these huge residual left pneumatoceles still displacing the mediastinal structures far to the right. Tube drainage was subsequently used effectively here because of further increase in size. Air leak resulted but closed in a few days.

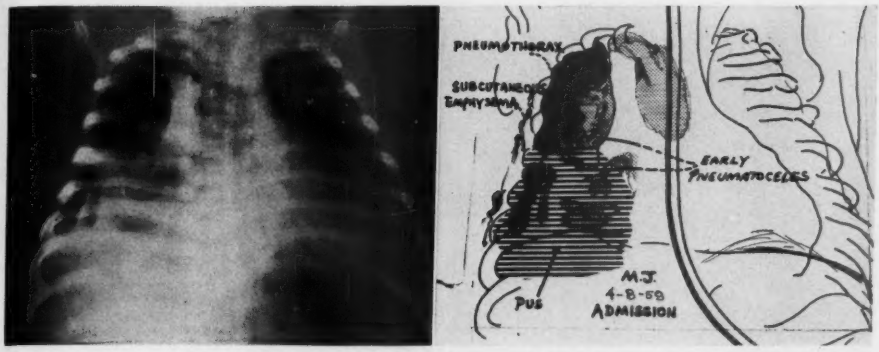


FIG. 6. Roentgenograms of a 2-week-old Indian infant transferred from another hospital with staphylococcal empyema persisting after thoracenteses. *A*. Pyopneumothorax present at admission. Note the subcutaneous emphysema which followed thoracentesis. Pus is somewhat loculated with air pockets entrapped in the thickened exudate. Mediastinum shifted left. Moderately severe distress.

FIG. 6*B*. Lateral view at admission showing multiple fluid levels and part of the fairly marked abdominal distension. Nasogastric tube is not quite far enough into the stomach to be maximally effective, a common finding unless each infant is specifically "measured" against his tube.

pneumothorax or the emphysematous pattern in the early phases of the disease. X-rays at frequent intervals early in the course of the disease can demonstrate the rapid change factor useful in diagnosis and even occasionally in predicting the imminent and possibly sudden manifestation of a complication. Lateral views are especially useful in permitting interpretation of the multiple densities and rarefactions encountered in the complicated case and are necessary to guide precise placement of drainage tubes. Recent x-ray films should, of course, be available for the surgeon when he inserts the chest tube, and it is particularly important to have immediate post-thoracostomy films to furnish precise information as to tube position, efficiency of drainage and especially lung re-expansion as well as the status of the previously obscured lung. Although the remarkably normal eventual appearance of the follow-up chest x-ray discourages further study, bronchograms of recovered patients should help to clarify the pathologic mechanisms.

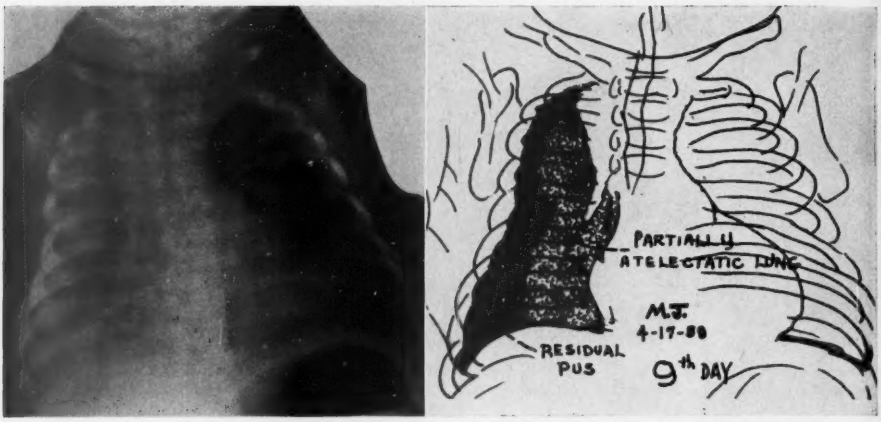


FIG. 6*C*. Ninth day. 1 day after first tube was prematurely removed. It had evacuated the air promptly along with enough of the pus to correct mediastinal shift. Extensive pulmonary infiltrative changes are evident even though a considerable amount of fluid remains. Without more adequate drainage this combination of empyema and partial atelectasis could have led to need for decortication. Tube was indicated here although it was apparently withheld because of satisfactory clinical course.

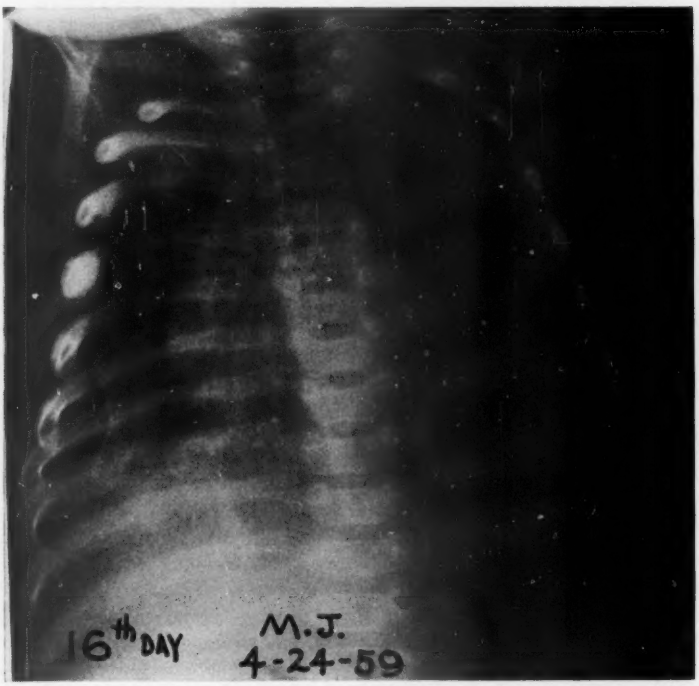


FIG. 6*D*. Sixteenth day. Continued resolution of pyothorax with increased amount of aerated lung, but residual "pleural thickening" and probably loculated pus at right apex.

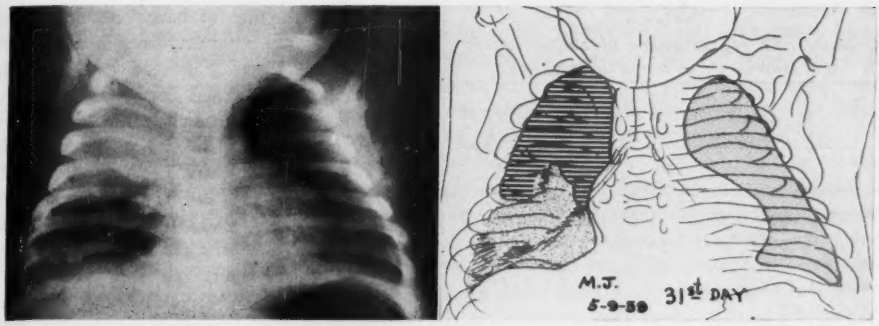


FIG. 6*E*. Thirty-first day. Residual apical pocket finally flared up, but tube was withheld here because of "dry tap." Fever persisted until tube drainage was reinstituted.

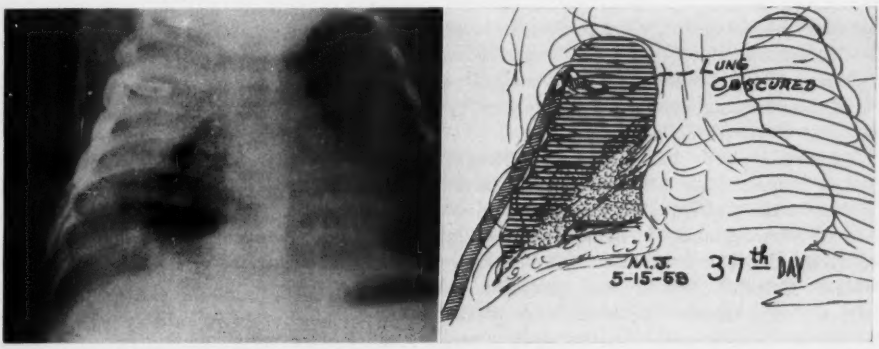


FIG. 6*F*. Thirty-seventh day. New tube in place. Marked residual pleuritis, but much better pulmonary expansion than the diagram suggests. Varidase instillations and irrigations seemed helpful here

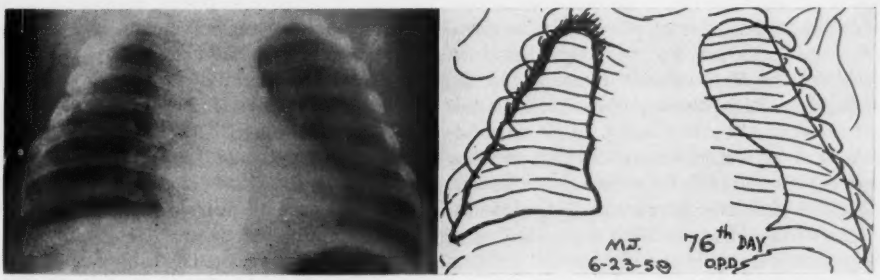


FIG. 6*G*. Seventy-sixth day. Progressive apical clearing has left only moderate residua pleural thickening here. Clinic follow-up film taken 4 months later was entirely normal.

SURGICAL CONSIDERATIONS

Our patients with staphylococcic pneumonia in Boston and Brooklyn encountered intrathoracic complications with a relative and absolute frequency similar to the approximate average of the reports of several recent authors[3, 16-19, 33, 36, 40] (table 2).

Treatment of intrathoracic complications may be considered under three general headings: antimicrobial, general supportive and drainage. The principle of using an appropriate drug in an adequate dosage has been stressed by the pediatricians directing the care of the infant with pneumonia. The development of complications is

TABLE 2

Intrathoracic complications of staphylococcic pneumonia—from recent publications

Complication	Percentage of Occurrence
	%
Empyema	70
Pneumatocele	50
Pyopneumothorax	
Simple	20
Tension	20
Fibrothorax	5
Abscess	1–2
Pericarditis	1–2

no reason for a change in the drug unless the most effective agent has been previously withheld for any reason. This has required adequate identification of the offending organism with guidance by sensitivity tests and clinical results.

I personally have had little indication to continue to have much faith in the use of penicillin, even in the massive doses used by its advocates. Several groups, including Ziegra[44] in Brooklyn and Cooke and Bennett[40] at The Johns Hopkins Hospital, believe that penicillin in massive doses remains a useful and potent bacteriostatic and even bacteriocidal drug, despite the low percentage of susceptible organisms as shown by the disc sensitivity method. In most of our cases the combination of chloramphenicol (75 to 100 mg. per kg. per day) and erythromycin (50 mg. per kg. per day) has been considered the most effective measure and has remained the initial therapy of choice. Concomitant administration is thought to be advantageous and the intravenous or intramuscular route is preferred initially, a change being made to the oral route only after a satisfactory clinical response. Riley[39] has found the increased absorption of erythromycin propionate to exert a favorable influence. The cases of Disney and co-workers,[13] studied by lung puncture technique, have recently provided evidence to support the policy of our pediatricians who have continued the specific therapy for 3 or 4 weeks after discharge from the hospital. Also, massive penicillin was usually included in the initial therapy plan in Brooklyn for the reason previously mentioned and because it was thought to provide better penetrance into the local lesion. Albamycin was used on occasion in Brooklyn in place of erythromycin, and the use of bacitracin systemically and topically appeared promising in the last few cases treated there. The studies of Austrian,[1] Rantz and Randall[37] and Perry[34] have indicated the effective antistaphylococcal qualities of bacitracin, and further trial of its use intrapleurally seems indicated. Pryles[36] has emphasized topical therapy and has used up to 10,000 units twice daily made up in isotonic saline solution with concentration of 500 to 1000 units units per ml. He has encountered no renal complications in a small series. Recently Oliver and Clatworthy[33] have made vigorous recommendations for a short 4- to 5-day course of intramuscular bacitracin given daily as 1000 to 1500 units per kg. in four divided doses, followed by a bacteriostatic drug suggested by the sensitivity tests (*e.g.*, erythromycin, 30 to 50 mg. per kg. per day) until the temperature has been normal for 2 weeks. They believed it to be particularly valuable in the infants under 1 month of age. They also warned of their experience with what they suspect to have been chloramphenicol toxicity in the young infants and caution against doses exceeding 50 mg. per kg.

On the newer drugs available, vancomycin seems the most promising and actually is probably our most effective antistaphylococcic agent in use today.[27]

SUPPORTIVE CARE

The importance of general supportive care of the infant with empyema cannot be overemphasized. Although the respiratory emergency precipitated in an already ill infant by the sudden development of a tension pyopneumothorax may demand immediate decompression as a life-saving measure, only vigorous and meticulous attention to the details of nonoperative care will allow the subsequent recovery of some of the severely ill patients.

The use of an oxygenated, humidified atmosphere is widely advocated, but it should be recognized that the need for high oxygen concentration may be extremely critical in the patient with very little remaining undiseased, unobstructed or uncompressed lung, with the increased metabolic needs of fever and infection, impaired diaphragmatic excursions due to abdominal distention and a low circulating hemoglobin capacity. Extra dividends of a cool mist environment are the antipyretic effectiveness of a cooled tent or incubator and possibly the decompressive

effect on intestinal gaseous distention if a very high oxygen content of the inspired air is achieved.[15] Hyperpyrexia and convulsions in children should be prevented by utilizing antipyretic drugs and more aggressive methods as indicated. The only residual ill effect personally observed in the survivors from the severe cases was in one infant who experienced permanent moderate central nervous system damage after several febrile convulsions associated with a period of apnea and anoxia. This must be considered a preventable complication. Simple nursing measures to avoid excessive environmental temperatures and clothing are usually effective in prevention. The established case needs vigorous measures which include discrete sedation in the older child, prompt heat dissipation by tepid surface sponges and electric fan and cold saline enemas. Perhaps the simplest and best method is one practically never seen outside the operating suite, the use of an ice-water mattress (Davol) or electric cooling blanket (Thermo-rite, etc.) The former is inexpensive and quite well adapted for infants. The observations of Williams and Spencer[43] on the prevention of permanent brain damage following cardiac arrest by induced hypothermia offer real hope for those who fail to obtain the benefits of prevention.

Maintenance of the circulating blood volume and composition is often not granted the attention that it deserves. The danger of overload by transfusion exists in cases with both respiratory and circulatory embarrassment, but the benefits of restoration of normal hemoglobin concentration are real. The use of repeated small transfusions or of packed red cells is an effective safety measure. Cardiac failure may ensue from the effects of anemia, toxemia, impaired venous return and even a sort of cor pulmonale. Digitalization may occasionally be beneficial and can be carried out promptly by the intravenous route.

The frequency of gastrointestinal symptoms has been commented on by previous authors[4, 16, 19, 21] and a primarily gaseous distention has been a striking feature in our more severe cases. This dysfunction is thought to be an ileus secondary to the acute intrathoracic disease, but peristaltic audibility has varied. Management is not easy and concentration should be on anticipation and control, if not prevention, by use of enemas, nasogastric intermittent suction, tube feeding, etc. Prompt complete evacuation of the pleural space with re-expansion of the lung will avoid many such troubles by prompting an earlier control of the infection and ventilatory defect. Diarrhea may precede, follow or substitute for the ileus, but is rarely more than a nuisance. Occasionally pain and vomiting accompany the distention and may lead to difficulty in diagnosis. One patient with these symptoms, seen in Boston, prompted a very early chest x-ray which led to our best documentation of the very rapid progression from near normal to a tension pyopneumothorax with dangerous mediastinal shift. Gavage feeding must be carefully controlled in the group with distention; aspiration deaths have been recorded, and diarrhea can be precipitated. A low Fowler's position, or at least elevation of the head of the crib 20 to 30°, is often helpful because of improved swallowing function and diaphragmatic excursion in the distended patients.

If the infant requires prolonged parenteral therapy, fluid and electrolyte imbalance can develop unless the intake and output and body weights are closely followed. Supplementary vitamins should be furnished via the prevailing route daily. Actual malnutrition has rarely been a problem because of the extreme rarity of the progression of a primary to a chronic stage. Clearly, oral alimentation is the best method, but occasionally plasma as well as blood or packed cells may prove beneficial in partial replacement of prolonged or excessive exudative losses and their sequelae.

DRAINAGE

The principles of surgical drainage of empyema in children[20, 26, 32] have changed little over the years, but the modifications of the empyema problem reflect the absolute and relative increase of the staphylococcic disease, as well as the potency of the newest antimicrobial and the drug resistance of the bacterial strain. Successful management of the complicated case of staphylococcic pneumonia in an infant requires that the pediatrician and the surgeon work in close cooperation. A primarily educational arrangement permitting our surgical residents in Brooklyn to be invited to follow cases of staphylococcic pneumonia in a stage prior to the onset of pleural complications proved, on several occasions, to expedite rapid and effective management of complications as they arose. Disciplinary prejudices dissolved in the presence of a critically ill patient, and there evolved an effective and preferred treatment plan which sought to provide quick control of pleural complication dangers along with vigorous sup-

portive care, thereby permitting the ultimate prognosis to approximate that of the underlying pneumonia. Since submission of the abstract of this presentation with its proposal that all physicians become acquainted with the simple and often life saving drainage procedures required for the treatment of this disease, Oliver and coworkers[33] have published an excellent critical review of their own results which led them to a similar conclusion. It should be the responsibility of the surgeon to initiate and perform or supervise the drainage procedure and, in particular, to teach the pediatrician the details of emergency intercostal tube insertion. There is no more effective method of emphasizing early diagnosis than to arrange participation in the acute and definitive drainage procedure for the group of physicians who will be best situated to anticipate the suddenly progressing disease stages.

During the past 6 years, our list of indications for drainage of staphylococcic empyema has become progressively shorter but broader. I now advocate closed thoracostomy tube drainage for even the apparently uncomplicated pyothorax in an infant. The procedure is especially simple as now practiced. It is safe and effective when properly performed. Most pediatric centers have gradually discarded the needle aspiration attempts at drainage as their experience has increased, because it has often proved ineffective, and indeed troublesome if diligently applied daily. Water-seal suction tube drainage provides adequate definitive drainage in almost every instance, and the old sequence, terminating in rib resection and semi-open tube drainage of the chronic cavity, is no longer seen except in a rare case of pneumococcic empyema. Closed chest drainage in the severe case provides help in several ways. (1) Prompt improvement in ventilation is often lifesaving; (2) evacuation of the exudate diminishes the systemic toxicity associated with undrained pus; (3) prompt re-expansion of the lung avoids the chance of chronicity, fibrothorax and subsequent need for decortication.

Workers in Columbus, Ohio[33] have used phagic typing of the staphylococcus for several years and have become so impressed with the association of certain strains (notably phagic type 80-81) with the more serious pleural developments that they have proposed a "close chart" and bedside availability of thoracentesis and thoracostomy sets when organisms with this type are isolated from even a remote source on the patient. Thoracentesis is a well established procedure, and consequently, adequate equipment and technical experience are widely available. Chest tube insertion is hardly more complicated, but paradoxically, a detailed presentation seems necessary to convince listeners of the simplicity. Because of my conviction that this life saving procedure should be mastered by all physicians, and because of a simplification of equipment and technique that has proved useful, the technique is here described and illustrated in practical detail.

CLOSED THORACOSTOMY

This technique is used to establish intercostal tube water-seal suction drainage. The use of a well equipped treatment room or the operating room is preferred so that contamination of the open ward can be minimized. The operator and assistants should be masked, gowned and gloved for mutual protection. One-half per cent procaine or Xylocaine is used for local infiltration anesthesia. If the set-up is familiar and equipment prearranged, one assistant to restrain the child will suffice. Recent posteroanterior and lateral chest x-rays should be available to guide a more precise placement. The upright position for all but the most prostrated patient has been used with support and restraint from the front, occasionally with a head-on-pillow position over a bed table. The lateral position is also satisfactory. If there is no loculation of air and fluid to direct the optimal tube site, a convenient point is chosen approximately in the midaxillary line in the fifth or sixth interspace (fig. 7). If a second catheter is inserted either electively at the first drainage or subsequently because of a specific indication such as loculation, the second interspace anteriorly is most commonly used. After skin preparation with ether and a tincture solution, a sterile towel can be advantageously draped transversely about the lower chest at the level selected for puncture and secured with adhesive at each end of the towel to the patient. Additional towels should be used to prepare a sterile flat area near the patient. The very ill infant will benefit from oxygen administered during the procedure, which may on occasion be most expeditiously performed in the patient's bed.

Skin and subcutaneous tissue are anesthetized with an intradermal needle which is followed, in all but the tiny infants, with a short bevel 22-

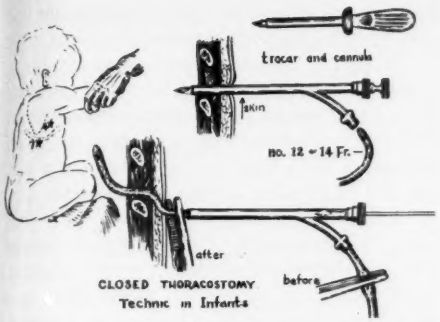
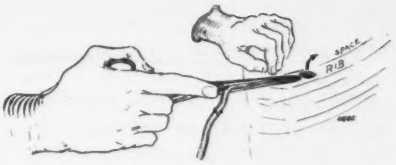


FIG. 7. Features of the trocar technique for closed thoracostomy drainage. The lower trocar (Oehsner bladder type) is preferable but requires careful matching of sizes with tubes. Available trocars should be sizes 10 to 16 French; catheters, sizes 10 to 12 French for infants, 14 to 16 French for children. (See text for other details.)

gauge needle down to the rib and then redirected upward through the intercostal space and beneath the endothoracic fascia, for injection of additional anesthetic solution.

A preferably new, straight, red rubber urethral catheter, 10 to 16 French according to patient size, has previously been provided with extra holes in the distal 3 cm. (removing not over one-third of tube circumference) and marked with a ligature or knife scratch or, if prior to sterilization, with a ballpoint pen at the skin level so as to limit insertion to 4 to 5 cm. into the intrapleural space. The commonly described technique is the trocar method which I first learned and used for several years. Because of difficulty in arranging for an adequate selection of trocars in a large hospital, in 1955 I chose to treat a very sick infant with a technique previously used for adults. It was effective and subsequently has proved so successful and simple that the trocar technique is rarely used now, even when proper equipment is at hand. This modification consists essentially of punching a hemostatic clamp (which grasps the catheter just back of the tip) through the deep chest wall layers into the pleural cavity (fig. 8). A 3- to 4-mm. skin incision is helpful and a narrow tunnel may be burrowed with a sharp curved hemostat down near, but not through, the pleura. Our preferred insertion clamp (fig. 9) is useful at 5½ to 7 in. in length with gently curved blades and semiblunt point preferred. A Bainbridge thyroid hemostatic forcep with the serrations ground down or a baby intestinal forcep (without



CLOSED INTERCOSTAL DRAINAGE OR TUBE THORACOSTOMY, PERFORMED WITHOUT TROCAR

FIG. 8. Simplified technique of intercostal tube insertion using a "controlled blunt punch."

rubber) will best release the inserted tube, but various standard clamps have proved satisfactory. This should grasp the catheter so as to almost reach the tip and should, if possible, occlude the side wall holes while the main lumen is clamped distally. The perforating thrust seems to require moderate force, especially at the physician's first experience, and the clamp should be held in such a way as to limit the depth of any sudden penetration. There may be a sudden gush of pus through, or even around, the catheter and clamp as the latter is released and withdrawn and the tube is threaded in to the appropriate mark. Culture material is ample to corroborate that usually acquired by needle aspiration earlier. Flexion of the infant's chest to prevent a convex surface provides slightly wider intercostal spaces and eases penetration by providing better resistance. The tube can be held in place by a hemostat until it is fixed to the skin and chest wall (fig. 10). A single suture can be used or, if the hole is quite snug, adhesive fixation (nonrestrictive type preferable) suffices with tincture of benzoin on the skin. Controlled-curve gauze support will prevent kinking at the chest wall and an extra tape attachment can be placed to protect the exit site from a sudden tug. The catheter is promptly aspirated with a large syringe if accurate volume measure is desired, or connected to a previously assembled three-bottle suction drainage system, usually with 10 to 12 cm. of water at negative pressure (rather than the small amount illustrated) which can be readily provided with a standard vacuum or even intermittent type pump (such as the Gomco thermotic). Very rarely an air leak will prove excessive and a pump capable of moving more air will be necessary. A T-tube metal faucet attachment has been effective when there were insufficient suction machines to provide for 7 infants simultaneously under treatment

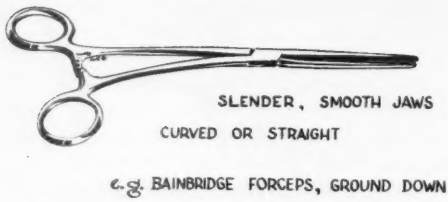


FIG. 9. Modified hemostat used for insertion of chest tube. Absence of serrations will aid withdrawal. Curved shape has proved preferable.

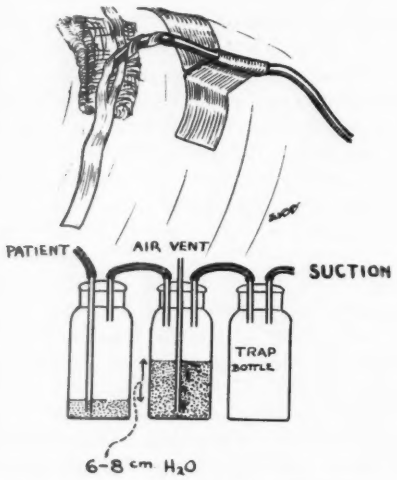


FIG. 10. *Upper:* Intercostal tube security as contributed by adhesive fixation to the chest wall. Additional tube-to-skin tape and an extra pad of gauze used to insure a gentle curve are not illustrated. *Lower:* Suction drainage bottle system with sterile water-seal collection bottle nearest the patient, air vent bottle next to regulate the vacuum suction applied to the patient, and the trap bottle illustrated because of its importance in protecting the pump. The effective suction is measured by the difference in length of underwater projections of the tubes in the first two bottles, and 10 to 12 cm. of H_2O are now used even for infants.

for staphylococcic empyema. Once connected, the bottles should be kept at least 2 feet below chest level and should be transported without clamping the tubing. Water-seal drainage without suction is relatively ineffective in small infants because of their limited tidal volume.

This simplified technique is admirably adapted to the occasional urgent situation presented by a severe tension pyopneumothorax and can even supplant the emergency needle decompression. The response of the patient to drainage is often dramatic, and a desperately ill, gasping infant will often be immediately tremendously relieved, with slower deeper respirations accompanied by a prompt and fairly deep, but relatively peaceful, sleep. On occasions, the apparent clinical benefit has far exceeded that expected from the small amount of fluid removed—a phenomenon also observed rarely after a single thoracentesis.

I find that currently we are more frequently using two tubes electively in cases with massive or unevenly distributed effusion. A second or even a third tube may be needed subsequently and should be inserted without hesitation. An immediate post-thoracostomy portable chest x-ray is important to show the tube position and may well show the lung already fully expanded. Repositioning of the tube may be safely carried out at any time indicated, keeping in mind the location of the perforations to avoid the potential subcutaneous emphysema that can result.

I have been best satisfied when the tubes were gently and daily irrigated for patency determination with a small volume of sterile saline using optimal precautions, a 30-cc. syringe and catheter adapter. Antibiotic solutions such as bacitracin can be instilled at regular intervals and Varidase instillations requiring periodic tube clamping may be used. Close observation is important in the latter group while the water-seal is clamped.

The duration of suction drainage has varied widely. The average in the Boston hospital was 13 days, the longest 33 days.[19] Our experience in Brooklyn was quite similar, the average being 14 days with a range of 6 to 30 days. Suction was continued until purulent drainage and air leak had not been evident for 3 to 4 days and the tube was usually removed promptly without temporary clamping. All bronchopleural fistulas in my experience have eventually closed with tube suction drainage, and this result is reported by others, although some surgeons seem to fear that vacuum will tend to keep such a fistula open.

On several occasions when the diagnosis was not clear, and on a few when the huge cystic lesion was thought to be a large pneumatocele, we have inserted the closed thoracostomy tube directly into this air space. No ill effects have been noted in these instances, and deflation of such areas has been expedited, although we have not encountered an early critical situation like that handled so effectively by Watkins with intracavitary suction drainage.[41] In no instance has a tension pneumothorax developed with a properly functioning

tube in place, but when irrigation patency was not checked frequently, partial loss of re-expansion has occurred several times. In some cases the pus has been viscous because of plastic and cellular debris and fibrin clots, with consequent decreased effectiveness of drainage. The instillation of Varidase solution has seemed to be useful then and may in 2 cases have prevented the need for decortication (fig. 6). Very few questionably related febrile episodes have occurred and I have not incriminated this procedure with any reopening of a bronchopleural fistula. It is difficult to accept the idea that the occasionally reported reopened air leak has been harmful or even necessarily related, since any really worthwhile healing process would be unlikely to regress significantly when bathed by the Varidase solution. Several cases in Boston, 7 in Brooklyn and 2 in Oklahoma City have been treated in this manner, generally by daily instillation of 10,000 to 20,000 units of Varidase in 20 to 30 cc. of sodium chloride solution through the tube, and clamping of the tube for 2 to 4 hours. Hendren[19] has reported 2 cases of moderate intrapleural bleeding in children whose platelet counts proved to be low. The topical use of neomycin has been avoided because of the danger of toxicity from rapid absorption via inflamed surfaces.

None of the cases personally encountered has required or received decortication, although the principles and techniques reviewed by Klassen[23] are sound. His indications for operative decortication in a rather surprisingly large series were: (1) fibrinofibrothorax (sterile), (2) unresolved loculated empyema, (3) persistent bronchopleural cutaneous fistula. His cases were predominantly of the late diagnosis type and occurred from 1946 to 1949. Four similarly late "transferred" patients were treated by decortication among the 75 cases reported from Boston Children's Hospital.[19] With early and adequate medical treatment of the pneumonia, reinforced by prompt recognition of complications and aggressive management of closed suction drainage therapy, most indications for these more extensive surgical procedures can be avoided. The exceptional tendency for spontaneous resolution of radiographic changes typical of exudate and thickened pleura prompts my general agreement with Lindskog[3] and Emerson[18] who "advise against unnecessarily early or emergency decortication or corrective pulmonary surgery." The latter phrase indicates resection and involves also the problems of pulmonary abscess, pneumatocele, localized emphysema and congenital cysts.[8, 12, 16, 31, 35] These are beyond the scope of my discussion, except for a confirmation of the infrequency of such lesions. The dramatic improvement of some of the few reported operated cases (such as the emergency lobectomy by Moore[31]) is a tribute to the surgical judgment that recognizes the occasional almost paradoxical desirability of a direct extirpative approach to a grossly infected unit of tissue.

The scoliosis formerly seen with chronic empyema in the growing child can be consistently avoided if prompt drainage prevents actual infection and fibrosis of the deeper chest wall tissues and intercostal muscles. The need for open drainage by rib resection should be encountered very rarely in the staphylococcic cases, although reliable methods for providing an air-tight seal have made it generally a more reliable procedure.

Staphylococcic pericarditis has been reported as an accompanying lesion and should be looked for, especially in the very severely ill, early case with a question of cardiac decompensation.

The differential diagnostic problems arising have not been rare and will be listed without description other than indication, by an asterisk, of those which might prompt unnecessary or misdirected operation: (1) acute abdominal condition,* (2) congenital diaphragmatic hernia,* (3) intrathoracic tumor* *versus* loculated empyema

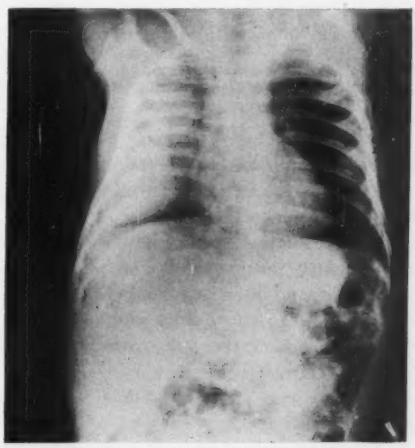


FIG. 11. Chest film of a 4-month-old infant with loculated interlobar empyema (*lower*) and an infiltrative lesion (*upper*) with mass present, with resulting differential diagnostic problem. Three days later the upper lesion was a typical thin-walled pneumatocele and the margins of the lower density were less distinct.

(fig. 11), (4) hematogenous or metastatic lung abscess, (5) cystic fibrosis of the pancreas with pulmonary disease (patchy atelectasis, emphysema, staphylococcus, occasional lobar atelectasis), and xanthomatous pulmonary infiltration with perforating pleuropulmonary lesion, (6) congenital irreversible bronchial cysts, (7) lobar emphysema, (8) other conditions which can be associated with pneumatoceles, such as Haemophilus influenzal pneumonia, β-streptococcic pneumonia, tuberculosis (acute suppurative phase of the bronchogenous type), hydrocarbon inhalation, pneumococcic pneumonia, and (9) other suppurative pneumonias which lack the pneumatocele forming tendency (Friedländer's bacillus, proteus organisms).

SUMMARY

A review of personal experiences with the treatment of complications of staphylococcic pneumonia in three separate geographic regions confirms the nationwide importance of this infection for both physician and surgeon.

Special attention is paid to pathologic sequences as keys to therapy based on a better understanding of the clinical and radiologic manifestations of the disease and its complications.

The importance of proper supportive care and surgical drainage are emphasized by a detailed presentation and discussion of techniques.

Primary staphylococcic pneumonia deserves the attention of pediatrician and surgeon alike for several reasons:

1. The relative and absolute increase in incidence is nationwide.

2. It is a distinct disease entity with complications the therapetuic implications of which are unlike those of the formerly encountered streptococcic and pneumococcic disease.

3. The surgical complications—empyema, pneumothorax, pneumatocele and fibrothorax—are frequent, and often early and critical.

4. The latter emergencies respond well to prompt and conservative but vigorous treatment, but the difference between a successful and a fatal outcome may still depend on a detail of intensive supportive care.

5. The nosocomial nature of many cases both demands and suggests preventive measures.

Closed thoracostomy suction drainage has proved to be an effective treatment method and certain departures from traditional use are now advocated:

1. It is indicated as soon as the diagnosis of staphylococcic empyema is made on a bacteriologic or radiologic basis.

2. Although drainage remains properly a surgical procedure, any physician caring for an infant with staphylococcic pneumonia should be prepared to institute emergency intercostal tube decompression for the occasional urgent tension pyopneumothorax.

3. A simplified trocarless method of intercostal tube thoracostomy is described and should be taught because of its effectiveness and universal ease of application.

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THE EFFECTS OF COMMONLY USED VASODILATORS ON THE BLOOD PRESSURE AND CONTRACTILE FORCE OF THE HEART[a]

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The clinical use of vasodilating drugs in peripheral vascular disease should be based upon knowledge of at least three factors: (1) The nature and extent of the vascular occlusion, *i.e.*, "mechanical" or spastic, and the degree of each component. This determines the potential vasoreactivity of the diseased portion of the vascular red. (2) The specific pharmacologic effect of the drugs employed upon the diseased portion of the vascular tree. For example, muscle vessels react differently to various drugs than do skin vessels. (3) The specific pharmacologic effect of the drugs upon the heart, *i.e.*, ultimate change in blood volume flow through a portion of the body obviously depends upon the volume of blood pumped and the caliber of the vessels through which the blood is pumped.

The authors have investigated the first factor listed above, studying available methods of evaluating the nature and extent of vascular occlusion, including the determination of the relative degree of mechanical occlusion and spasm in various types of vascular disease.[9] The second factor above has been studied by an attempt to determine the pharmacologic responses of the peripheral vessels in humans to four popular vasodilator drugs.[10] This present report primarily is concerned with the pharmacologic effects of the same four clinically popular vasodilator drugs upon the myocardium, to some extent relating these effects to those acting directly upon the vasomotor system.

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Presented at the Annual Meeting of the Southeastern Surgical Congress, New Orleans, Louisiana, 1960. Winner of the First Prize in Scientific Paper Award Contest. (Added in proof—Since his paper was submitted in June 1960, a subsequent paper has been published by members of our group.)

METHODS

Mongrel dogs were anesthetized with sodium Pentothal and open thoracotomy performed through a right fourth intercostal space incision. In each case, a polyethylene cannula was introduced into the femoral artery at the groin, and threaded up into the abdominal aorta for blood pressure measurement. A similar cannula was inserted into the femoral vein. A Walton-Brodie strain-gauge arch was sutured directly onto the musculature of the right ventricle.[1, 8] Electrocardiographic leads were placed onto the four extremities. Blood pressure was recorded by a Statham pressure transducer, simultaneously with contractile force curves on a Sanborn multichannel oscillographic recorder. After adjusting and calibrating the recording equipment, a tracing of the contractile force, blood pressure, and electrocardiogram (EKG) were made under control conditions. One of the following procedures was then carried out:

Procedure I. An infusion of each of the drugs to be tested was given intravenously using a dose equal to 1 mg. per kg. of body weight. The responses of the myocardial contractile force, blood pressure, and EKG to intravenous infusion of each of the following drugs were measured: azapetine hydrochloride (Ilidar), nylidrin hydrochloride (Arlidin), benzyl-imidazoline hydrochloride (Priscoline), and phenoxyisopropylnorsuprifen (Vasodilan). These drugs have been described and identified in a previous report.[10]

Procedure II. A "test dose" of nitroglycerine (0.1 mg. per kg. of body weight) was administered intravenously, and after waiting a suitable interim for the return to control levels, the intravenous infusion of each of the vasodilating drugs was carried out and the responses measured.

Procedure III. Procedure II was modified so that preceding the infusion of the vasodilator, a sympathetic block was done bilaterally, either with local infiltration of the chain and ganglia in the chest with 1 per cent Procaine solution or by chemical sympathetic blockade with a proven ganglionic blocking agent. The efficacy of the

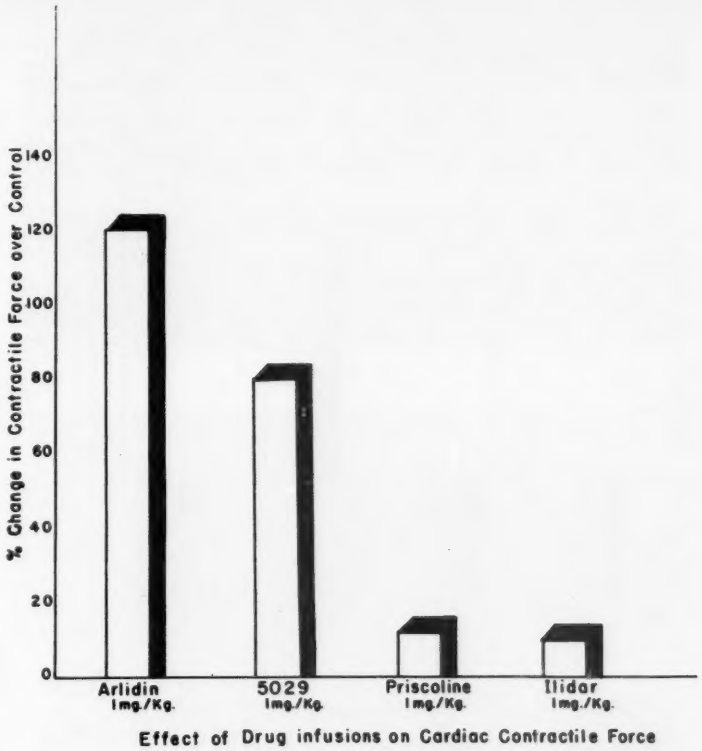

FIG. 1

ganglionic blockade was demonstrated in each case by failure of the typical test pattern of hypotension and myocardial contractility stimulation after the standard dose of nitroglycerine (see discussion).

Procedure IV. Procedure II was modified so that infusions of one of the vasodilating drugs were given following the infusion of another of the drugs, in order to attempt to elicit any synergistic, additive, or antagonistic effects. In earlier experiments, a small infusion of the vasodilating agent diluted to 150 cc. with normal saline was utilized as the test solution. Later the drug was given as a single rapid injection. No statistical difference between these methods of administration was observed in the experiments.

RESULTS

A summary of the results is graphically presented in figures 1 and 2. The average values represented in these two illustrations were derived from data obtained in experiments performed upon 32 animals. In figure 1, the effects of the various drug infusions upon myocardial intrinsic contractile force are compared. Identical doses of Arlidin and Vasodilan produce rather marked stimulation of cardiac contractile force, while Priscoline and Ilidar exert little effect upon the myocardium. Figure 2 demonstrates the comparative changes produced in the blood pressure following the administration of identical doses of the four drugs. Blood pressure changes are expressed in the graph as percentage of change in the control value.

DISCUSSION

The results of these experiments indicate that the total resultant increase of peripheral blood flow produced by a vasodilating drug depends upon at least two mechanisms. One of these mechanisms is dilatation of the peripheral vascular bed, and the other is an increase in the

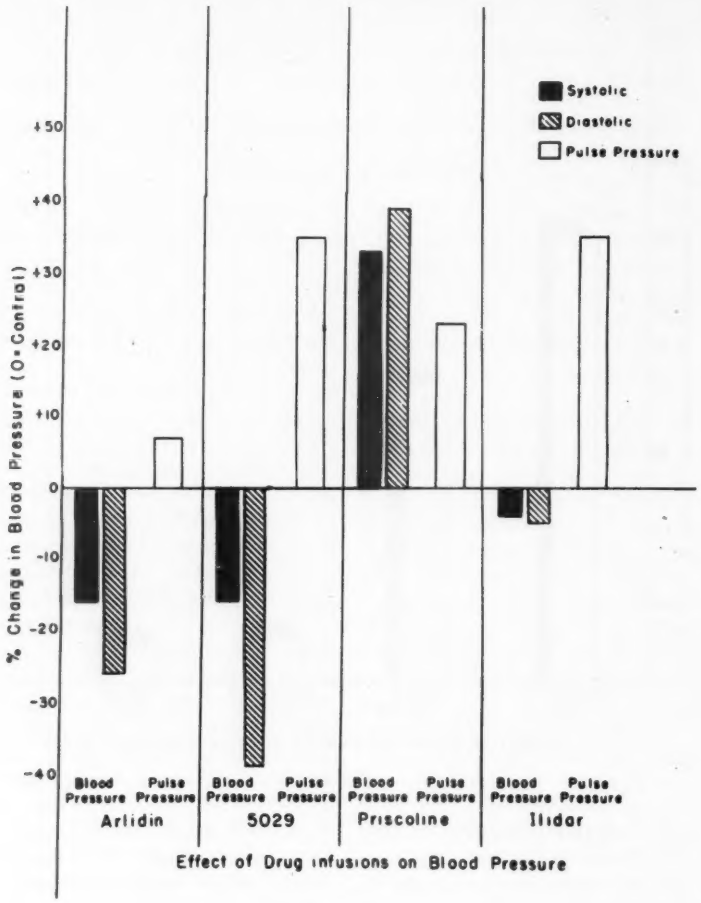

FIG. 2

amount of blood pumped per cardiac cycle by the heart, due to an increase in myocardial contractility. In order to determine whether the stimulating action of these drugs upon the heart force is a direct effect of the drug upon the myocardium, or an indirect effect created through "reflex" phenomena, further analysis of the data is necessary. Representative tracings taken from these experiments are used in the following discussion to illustrate average findings.

Figure 3 shows the administration of Ilidar in a dose of 1 mg. per kg. of body weight. The top tracing represents blood pressure, and the bottom represents contractile force. The EKG recording is not illustrated because there were no significant electrocardiographic changes following the administration of Ilidar in these experiments. The infusion produced a slight and transient drop in both the systolic and diastolic blood pressure initially, followed by a short period of bradycardia, followed in turn by an increase in blood pressure, both systolic and diastolic. After the transient hypotension, a brief decrease in contractile force occurred. This in turn was followed by a progressive rise in the contractile force to a level of about 150 to 155 per cent of the control figure, representing an increase of approximately 50 per cent. This increase was delayed, following the initial blood pressure drop. This pattern of a delayed increase in contractile force following a

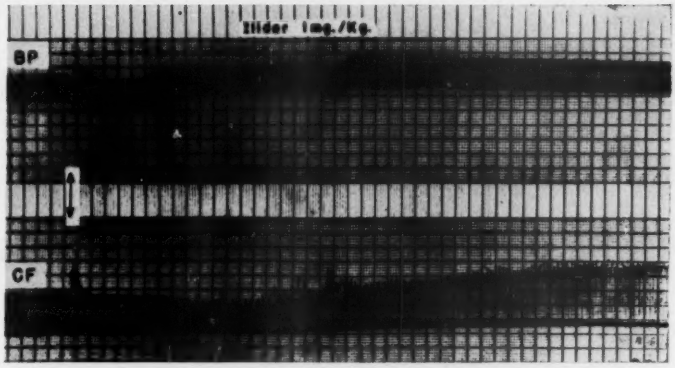

FIG. 3

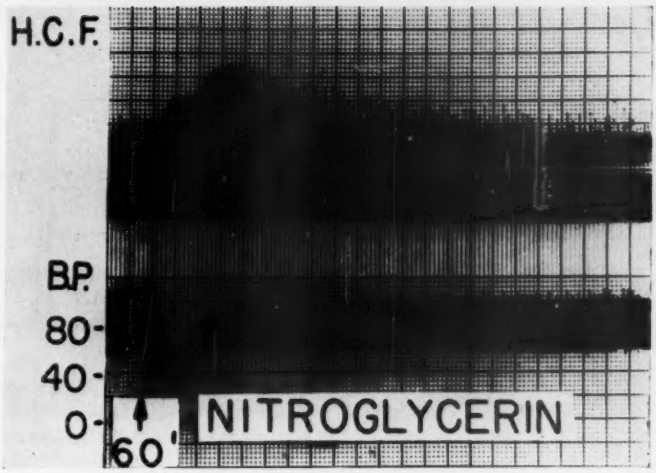

FIG. 4

transient hypotension has been previously noted to occur as a reflex phenomenon resulting from a release of catechol amines, initiated on a sympathetic level by transient hypotension.[5]

Darby *et al.*[5] have demonstrated this phenomenon, utilizing vasodepressor drugs such as quinidine and nitroglycerine, measuring an increase in heart contractile force of approximately 30 per cent above control levels in anesthetized dogs (fig. 4). The marked increase in contractility (top tracing) which follows the administration of nitroglycerine in the unblocked anesthetized dog is seen to approximate a 50 to 55 per cent increase in this case. They further demonstrated that after a sympathetic block the administration of these substances elicits no consistently significant change in heart force. The efficacy of a bilateral sympathetic block with Procaine in eliminating the reflex increase in contractile force produced by catechol amines release following the administration of nitroglycerine in a standard dose of 0.1 mg. per kg. is depicted in figure 5. The middle tracing shows a rather marked drop in blood pressure, the bottom tracing showing only a slight decrease in contractile force following the blood pressure drop, presumably representing myocardial depression due to a decreased coronary flow.

Infusions of Ilidar following a bilateral sympathetic block resulted in no change in contractile force, although an initial and transient slight drop in blood pressure occurred such as in figure 3.

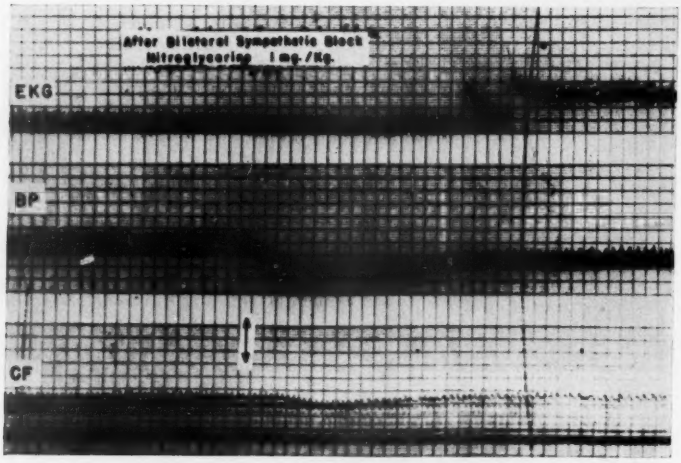

FIG. 5

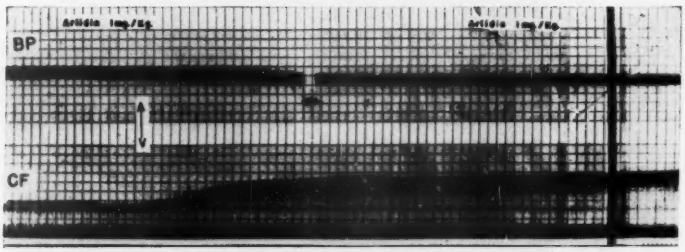

FIG. 6

Utilizing this same technique of infusion, sympathetic block, and repeat infusion, in other dogs with Priscoline, no alteration in the myocardial contractile force was detected. There was no consistently significant change in the peripheral blood pressure, following the administration of Priscoline at a dose of 1 mg. per kg. in the animals which had sympathetic blocks.

On the other hand, Arlidin in a dosage range of 1 mg. per kg., produces a rather marked myocardial stimulation (fig. 6). In this case, there was practically no change in the blood pressure, although myocardial contractility increased to almost 200 per cent of the control level. This degree of myocardial stimulation approaches the range of stimulation which occurs with the administration of a standard test dose of norepinephrine (2 μg. per kg.).[6] The increase in myocardial contractile force which occurred following Arlidin infusion was shown to be a positive inotropic effect, rather than a reflex phenomenon, since the increase in myocardial contractility persisted after sympathetic blockade with Procaine as described above. A similar response is seen following the administration of Vasodilan (fig. 7). In this case, there was a marked initial drop in diastolic blood pressure, although the systolic pressure and the contractile force increased. This suggests a significant increase in stroke volume, cardiac output, and probably is associated with elevated levels of myocardial metabolism. In general, Vasodilan produced somewhat less myocardial stimulation than did Arlidin in an identical dosage. However, both of these drugs produced the same general range of stimulation which occurred with the known potent myocardial stimulants such as epinephrine and norepinephrine. In figure 8, the stimulating effect of Vasodilan on the myocardium is seen to be a direct action of the drug, since the heart contractile force increase occurs following a bilateral sympathetic block. The

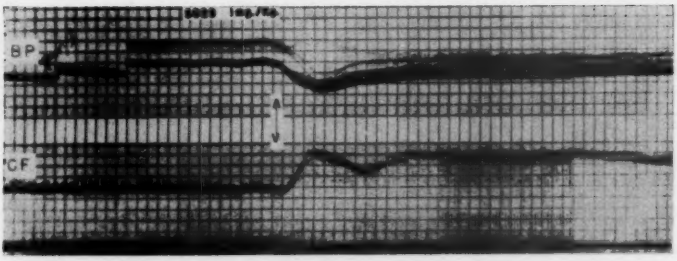

FIG. 7

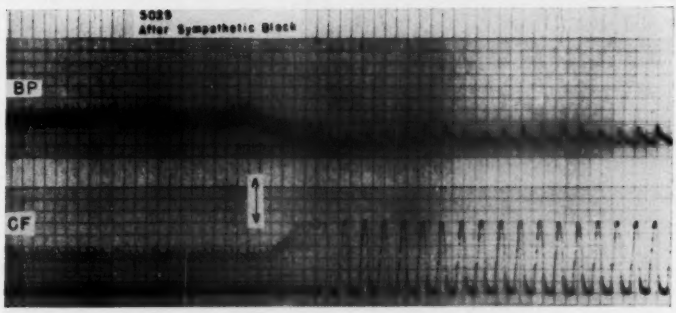

FIG. 8

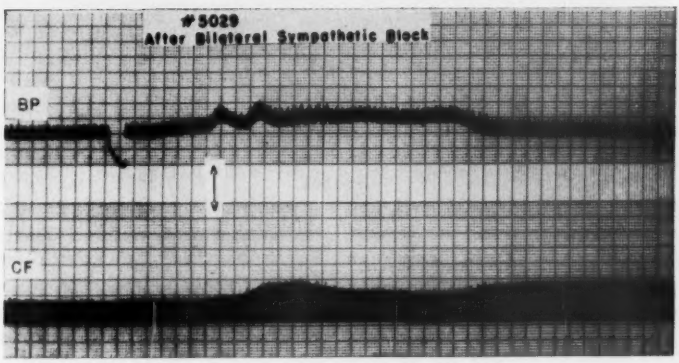

FIG. 9

degree of direct myocardial stimulation is less than in the unblocked animal, perhaps partly due to a more persistent hypotension.

The contractile force quantity following this infusion is therefore interpreted as being a resultant of three factors in the unblocked dog: (1) The direct stimulation of the myocardium by the drug, (2) possibly some initial diminution of contractile force due to diminution of the coronary blood flow following hypotension, and (3) stimulation of the myocardium by catechol amines reflexly released by the sympathetic nervous system in the hypotensive state. Figure 9 illustrates however, that the direct effect of the drug on the myocardium is the most prominent one.

Both Arlidin and Vasodilan were found to possess inotropic actions similar to epinephrine and norepinephrine in these studies. Figure 9 represents an infusion of Vasodilan after a sympathetic block. The contractile force was low during the control period, averaging only about

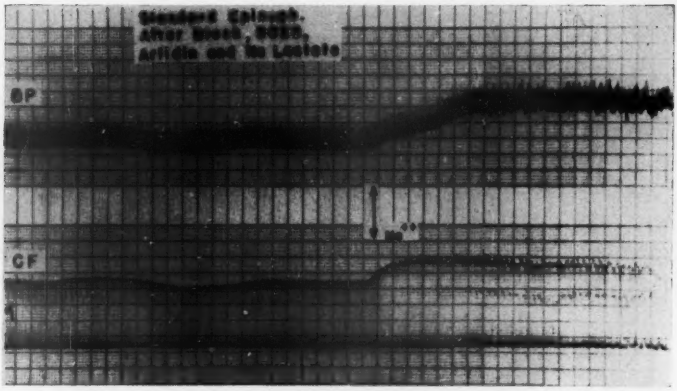

FIG. 10

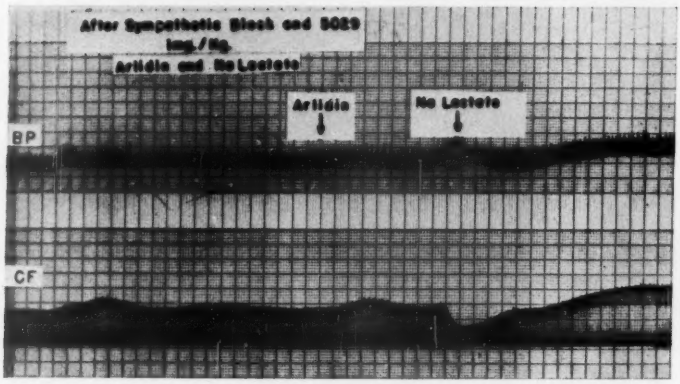

FIG. 11

50 per cent of the expected normal level (approximately 40 gm. of force). While the response to the drug infusion here is again in the range of 150 to 160 per cent increase, the quantitative degree of increase in stimulation is relatively slight. This same type of limited response in animals is noted following the administration of norepinephrine when the animal has been in a chronic state of shock, or when some situation has been imposed on the animal to produce a relative tissue acidosis.[3, 4, 7, 8, 11] The responsiveness of the cardiovascular mechanism to stimulation by adrenaline-like drugs can be restored by the elimination of the acidosis.[3, 7, 8] This was illustrated in figure 10, by allowing the animal to reach a point of acidosis in which no responsiveness to Vasodilan, Arlidin, or epinephrine could be demonstrated. Sodium lactate was then given intravenously, and a standard dose (2 minims) of epinephrine was given. Although this dose had previously exerted no effect upon the contractile force of blood pressure in the hypotensive acidotic animal, here a significant degree of myocardial stimulation and increase in blood pressure was produced. This is again illustrated in figure 11. Arlidin given intravenously caused no significant change in contractile force or blood pressure in a dog allowed to become acidotic, and in addition no synergistic or additive effect could be detected after the administration of Vasodilan. However, following the administration of sodium lactate, the positive inotropic effect of the previously administered drug is seen to emerge. The possibility of reflex stimulation by sympathoadrenal compensatory activity was eliminated in this type of experiment also by bilateral sympathetic block.

Figure 12 demonstrates a further similarity

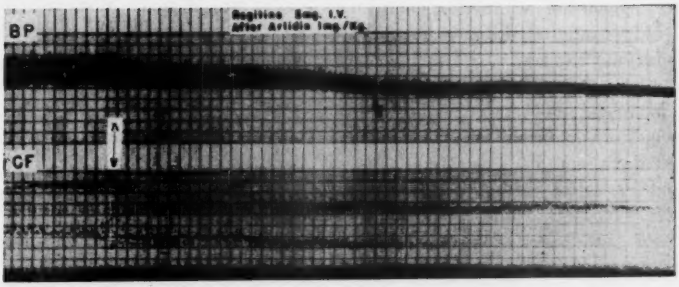

FIG. 12

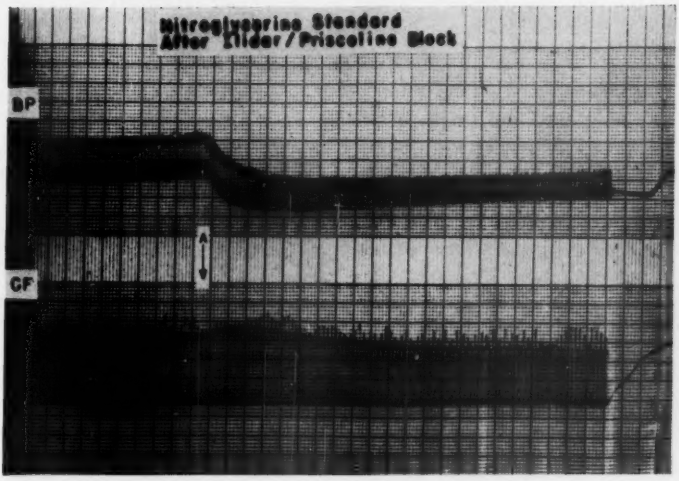

FIG. 13

between Arlidin or Vasodilan and epinephrine. The 30 per cent decrease in contractile force and 30 to 40 per cent decrease in blood pressure following the intravenous administration of Regitine in an animal previously stimulated by an intravenous infusion of Arlidin demonstrates that the adrenolytic agent Regitine antagonizes or nullifies the pharmacologic actions of Arlidin (and Vasodilan).

Figure 13 demonstrates that the administration of Ilidar or Priscoline in a dosage of 1 mg. per kg. of body weight eliminates the usual sympathoadrenal reflex myocardial stimulation which ordinarily follows an injection of a "test" dose of nitroglycerine. This suggests that these two drugs exert an effective ganglionic blocking activity. This has been previously confirmed in the case of Ilidar by Cotten *et al.*[2]

SUMMARY

1. Both Arlidin and Vasodilan exerted a potent stimulating effect upon the heart, and a vasodepressor effect on peripheral vasculature, creating a physiologic situation conceivably deleterious to a diseased myocardium. Partial protection from this effect may be afforded by vasopressor stimulation produced by the adrenaline-like substances which are reflexly released following the vasodepression which these drugs provoke.

2. Arlidin and Vasodilan pharmacologically simulate the actions of norepinephrine and epinephrine on the myocardium, but upon the peripheral vascular bed, their actions are basically vasodepressive and hypotensive.

3. Ilidar and Priscoline exert no direct stimulating effect upon the myocardium, and only a

minor over-all myocardial stimulation is produced, presumably mediated through a sympathoadrenal reflex mechanism.

4. The vasodilator drugs tested demonstrated no synergistic or additive effects upon the heart in similar dosages. In addition, no antagonistic effects between them were demonstrated.

5. Ilidar and Priscoline, in the dosages employed, effectively blocked certain parameters of sympathoadrenal function which ordinarily cause a stimulation of heart contractile force under conditions of hypotension.

6. The stimulating actions of Arlidan and Vasodilan upon the heart are obviated by the presence of acidosis, restored by the administration of a basic solution, and are antagonized by the administration of the adrenolytic agent Regitine.

Acknowledgments. The authors wish to express their appreciation for the advice and assistance of Dr. T. D. Darby, Department of Pharmacology, and Mr. and Mrs. R. A. Brown, Department of Medical Illustration.

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THE ROLE OF THORACOTOMY AND PARIETAL PLEURECTOMY IN THE DIAGNOSIS AND TREATMENT OF PLEURAL EFFUSIONS

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Pleural effusion frequently presents problems in both diagnosis and treatment. Statistically, the three most common causes of pleural effusion are neoplasms, congestive heart failure, and inflammatory lesions. Miscellaneous causes include pulmonary infarction, trauma, spontaneous pneumothorax, cirrhosis, subdiaphragmatic abscess, collagen diseases, chronic nephritis, and Meig's syndrome. From table 1, it can be seen that in a comparable series of over 400 cases of pleural effusion, reported a decade apart, those of indeterminate etiology decreased from 38 per cent to 17 per cent. This reduction in the incidence of indeterminate cases over the 10-year period, 1945–1955, is accounted for by an increased incidence of diagnosed malignancies. Nonetheless, one out of every six cases of pleural effusion encountered in the more recent series was without a definitive diagnosis.

When a pleural effusion is encountered in a younger person, it is initially presumed to be tuberculous. When encountered in a patient past the age of 45, malignancy is usually a prime consideration. Obviously, it is important to identify or exclude when possible tuberculosis as the cause of the effusion before specific therapy be given. To label an effusion as tuberculous without bacteriologic confirmation may commit the patient to a minimum of a year of intensive and needless therapy. The specific gravity and the cell count of the pleural effusion shed little or no light on the etiology. Of greater significance is the finding of bloody fluid, which in the majority of cases is due to malignancy.[6]

In an effort to increase the accuracy of diagnosis, Heller *et al.*,[5] in 1956 described the use of a Vim-Silverman needle in obtaining a biopsy of the parietal pleura. In their reported series of 20 cases, the procedure was diagnostic in only 9 cases. Breckler[1] abandoned this technique in favor of a small open thoracotomy which in his hands was no more hazardous and the tissue specimens were superior. He reported 16 cases biopsied in this manner with a resulting diagnosis in two-thirds of them. Sutliff[8] somewhat earlier described pleural biopsy using open thoracotomy performed under local anesthesia. He reported 21 cases. Of these, a diagnosis was obtained in all but one case, which was later proved to be carcinoma involving the pleura. From these and other[3] reported experiences it would appear that open thoracotomy in selected cases of pleural effusion offers the best method of obtaining a diagnosis when simpler diagnostic procedures have failed.

Since many of these cases of pleural effusion in this etiologically indeterminate group recur after repeated thoracenteses, some procedure which would effectively prevent re-accummulation of fluid would be welcomed by both the patient and the physician. Several methods which entail open thoracotomy have been employed to obliterate the pleural space and thereby prevent recurrent effusion. The one method with which we have had a limited experience is that of parietal pleurectomy. Besides obliterating the pleural space from the resulting adherence of the visceral pleura and chest wall surfaces, the resected specimen of pleura provides tissue for biopsy and cultures if indicated.

MATERIAL AND RESULTS

The following 7 cases, briefly reported, represent our experience with open thoracotomy and parietal pleurectomy in attempts to: (1) establish diagnosis, (2) eliminate recurrent pleural effusion. These represent selected cases of pleural effusion and the method of selection will be dealt with more fully under "Discussion."

Case 1. A 63-year-old white woman was admitted to the hospital complaining of cough, shortness of breath, and weight-loss. A left pleural effusion was diagnosed. Repeated cytologic examinations of the fluid were negative, as were the tuberculin tests. Culture of the pleural fluid

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TABLE 1

Statistical comparisons of diagnoses in two large groups of cases of pleural effusion reported a decade apart[8, 9]

Statistical Study of Pleural Effusion	1945, 444 Cases	1955, 463 Cases
	%	%
Indeterminate	38	17
Malignancy	38	53
(Cytology positive)	(30)	(42)
Congestive failure	9	10
Inflammatory	9	8
Miscellaneous	6	12

yielded *Candida albicans* on three occasions. Bronchoscopic examination was within normal limits, and a biopsy of an enlarged axillary lymph node revealed no malignancy. Because of the persistence of the pleural effusion, she was explored through a small left thoracotomy. She was found to have a collapse of the left lower lobe, and the incision was converted into a standard posterolateral thoracotomy. On removal of approximately 2000 cc. of serous fluid, and with the administration of positive pressure from the anesthetic bag, the lower lobe was easily inflated. Dissection of the lower lobe bronchus revealed no abnormality and careful inspection of the lung revealed no parenchymal lesion present. The pleura, other than being slightly thickened, did not appear unusual. By blunt dissection, the parietal pleura was freed from the endothoracic fascia, starting at the thoracotomy incision and extending superiorly over the apex of the thorax to the level of the aortic arch. The pleura was then freed and excised inferiorly, anteriorly, and posteriorly from the chest wall. No attempt was made to excise the diaphragmatic or pericardial aspects of the parietal pleura. Two fenestrated catheters were brought out through separate stab wounds for attachment to constant negative pressure in order to maintain the lung in full expansion. She made an uneventful postoperative convalescence and at no time has she developed a recurrence of the effusion. The pathologic diagnosis was subacute pleuritis. She has now been followed 5 years postoperatively. She has gained 20 pounds in weight, has no cough or shortness of breath, and has an essentially normal-appearing chest x-ray (fig. 1).

Case 2. A 66-year-old white woman was admitted to the hospital complaining of right anterior chest pain, shortness of breath, and cough. A right pleural effusion was diagnosed, and on three occasions 1000 to 1500 cc. of blood-tinged fluid was obtained by thoracentesis. Cytologic and bacteriologic examinations of the fluid were negative. Bronchoscopic examination, upper gastrointestinal and colon x-rays were within normal limits. Exploration through a right thoracotomy revealed the pleura to be studded with firm, yellowish-gray nodules grossly malignant. In some areas, the pleura was 2 cm. in thickness. Careful examination of the lung itself revealed no obvious primary lesion. Because of her apparent good health, palliative parietal pleurectomy was carried out. She made an uneventful postoperative convalescence. Pathologic diagnosis of the resected specimen of pleura was that of metastatic adenocarcinoma, primary site unknown. She remained asymptomatic for 22 months, and died 26 months postoperatively of carcinomatosis. (Necropsy was not obtained).

Case 3. A 48-year-old white woman was admitted to the hospital complaining of anterior chest pain, shortness of breath, and cough. A right pleural effusion was diagnosed by x-ray. Routine tuberculosis and fungus cultures were negative on three occasions, as was the cytologic examination of the straw-colored fluid obtained by thoracentesis. The tuberculin test was positive, but numerous cultures of sputum and gastric content were negative. Bronchoscopic examination was within normal limits. Because of the indeterminate nature of the effusion, the reluctance to place her on a year of antimicrobial therapy empirically, and the relative frequency of malignancy at her age, she was subjected to thoracotomy. On mobilizing the parietal pleura from the apex toward the azygos vein, numerous tubercles were seen studding the pleura. This area was resected with the remainder of the parietal pleura. No parenchymal lung lesion could be demonstrated. The pathologic diagnosis was that of a caseating granuloma. The specific diagnosis of tuberculosis, obtained by culturing the pleural tubercles, was not obtained until 1 month postoperatively. She had, however, been placed on antimicrobial therapy prior to surgery, and this was continued for 1 year. At this time, 2 years postoperatively, she is well, has had no further effusion, and no evidence of active tuberculosis (fig. 2).

Case 4. A 56-year-old white man with an extensive left pleural effusion requiring repeated thoracenteses was admitted to the hospital for evaluation. Bronchoscopy, supraclavicular lymph node biopsy, cultures and cytologic examinations of the pleural fluid failed to reveal the etiology. Because of the indeterminate etiology and the necessity for frequent thoracenteses, a left exploratory thoracotomy and parietal pleurectomy

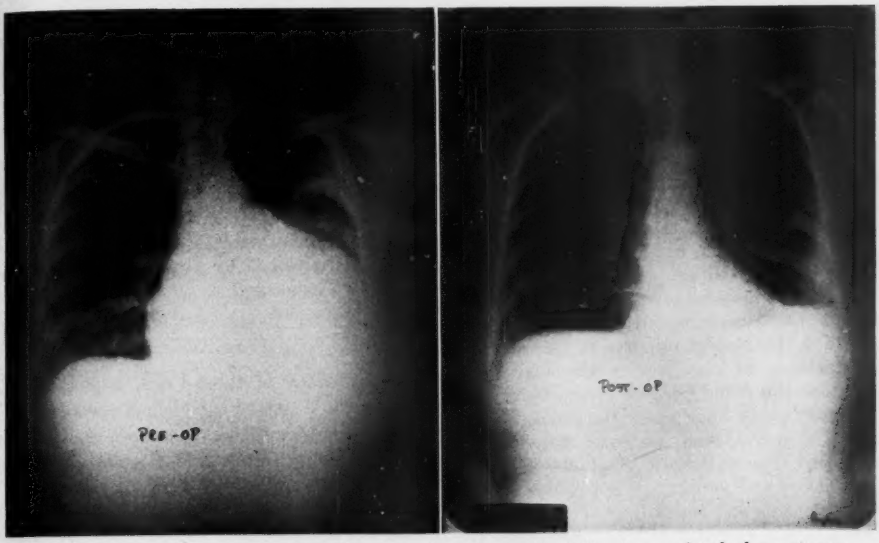

FIG. 1. Case 1, the postoperative film was taken 37 months following parietal pleurectomy

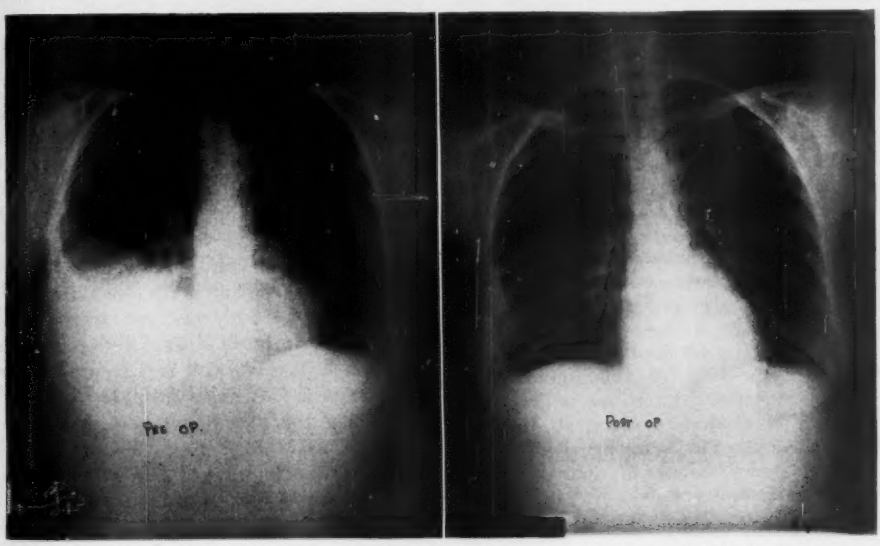

FIG. 2. Case 3, the postoperative film was taken 4 months following parietal pleurectomy

were performed. Pathologic examination of the resected pleura revealed only sheets of lymphocytes. He remained well for 5 months, and then went rapidly downhill and expired. Necropsy examination revealed a diffuse lymphosarcoma.

Case 5. A 68-year-old white female with a right pleural effusion was admitted to the hospital for evaluation. Ten years previously, she had had a right radical mastectomy. Although the effusion was presumably due to carcinoma metastatic from the breast, cytologic examination failed to reveal malignant cells. Because of her good general health, reasonable life expectancy, and because of the necessity for frequent thoracenteses, a right thoracotomy and parietal pleurectomy were performed. Pathologic examination of the resected

pleura revealed metastatic adenocarcinoma consistent with primary carcinoma of the breast. She remained well for 18 months postoperatively, at which time she was lost to further follow-up.

Case 6. A 47-year-old white man was admitted to the hospital for evaluation because of recurrent left pleural effusion over the period of the past 4 months. A comprehensive evaluation including liver and kidney function tests was within normal limits. A tuberculin test was positive, but the tubercle bacillus could not be identified either in the pleural fluid, sputum, or gastric aspirate. He was placed empirically on streptomycin and isoniazid for 3 months without improvement. Because of this lack of response to therapy, and the reluctance to keep him on this regimen, a left thoracotomy and parietal pleurectomy were performed. Other than some thickening of the pleura and pericardium, no gross abnormalities were noted. The pathologic diagnosis of the resected pleura and pericardial biopsy was chronic fibrous pleuritis and pericarditis. Portions of the resected pleura were cultured for tuberculosis and fungi. No growth was obtained. At this time, 25 months postoperatively, he is well and has had no further re-accummulation of fluid.

Case 7. A 54-year-old white female was admitted to the hospital because of a right pleural effusion and shortness of breath. She had had a left radical mastectomy 8 years, and a subsequent right radical mastectomy 2 years before this admission. Three thoracenteses in the course of 1 month yielded over 2 liters of serosanguinous fluid on each occasion. Cytologic examination revealed malignant cells. Bronchoscopic examination and x-rays of the spine were normal. Physical examination failed to reveal evidence of metastases elsewhere. Because her general condition was considered reasonably good, and because there existed an 8-year period since her first encounter with breast carcinoma, she was subjected to parietal pleurectomy with the expectation of 1 or more years of comfortable survival. Unfortunately, successful palliation lasted only 3 months, when generalized metastases became evident and she expired 5 months postoperatively.

DISCUSSION

Although at first glance it might appear that parietal pleurectomy is a radical form of treatment for pleural effusion, we wish to emphasize that the procedure is intended only for selected cases. Selection is based on a careful medical evaluation which suggests a reasonable life expectancy in a patient incapacitated by recurring pleural effusion and necessitating frequent thoracenteses for symptomatic relief. Furthermore, in many of these cases, a definitive diagnosis could not be established preoperatively. Since open thoracotomy has been shown to be often necessary to obtain a diagnosis, treatment of the effusion by parietal pleurectomy seems a reasonable procedure to add.

TABLE 2

Summary of cases reported (see text)

Case	Diagnosis	Follow-up Time	Status
		mo.	
1	Inflammatory?	60	Well
2	Malignant	26	Dead
3	Inflammatory (T.B.)	24	Well
4	Malignant	5	Dead
5	Malignant	18	Well
6	Inflammatory	25	Well
7	Malignant	5	Dead

In this small series of 7 cases, there has been no mortality, and the morbidity is that associated with the usual thoracotomy. Although extensive bleeding from the raw surface left by stripping and excising the parietal pleura is a distinct possibility, this has not been a complication in this series. This procedure is essentially the same procedure as that frequently used for the control of recurrent spontaneous pneumothorax. In large series of cases reported, it is noted that there is a satisfactorily low mortality and morbidity, and pulmonary function tests postoperatively have revealed no significant loss of respiratory function due to the procedure.

Parietal pleurectomy has been employed by others in the treatment of pleural effusion due to malignancy. Waterman *et al.*[10] reported favorable results in 5 cases. Two other methods of obliterating the pleural space to prevent recurrent effusion have been advocated. Both produce inflammatory irritation of the pleural surfaces, one by wiping with dry gauze,[7] the other by applying talc as the irritant.[2, 4] With subsequent re-expansion of the lung, synechiae form between the opposed inflamed pleural surfaces with resultant symphysis. The use of talc has recently been reported in 19 cases of malignant pleural effusion.[4] The average hospital stay in these cases was 10 days. In our 7 cases, the average hospital stay was 12 days, the longest being 20 and the shortest, 8 days. Although talc poudrage is a less extensive procedure, its failure to accomplish pleural sym-

physis has been reported in cases of recurrent pneumothorax, and for this reason we have not favored its use. All of these methods are dependent for success upon prompt re-expansion of the lung which must be maintained from 48 to 72 hours by adequate negative pressure applied to the drainage tubes from the chest.

Two of our seven cases represent failure, as both obtained little if any palliation and both succumbed in less than 6 months (table 2). These two disappointing results represent failure of proper selection of cases for the procedure. In none of the cases, however, was there a recurrence of the pleural effusion.

SUMMARY

There are many causes, systemic as well as local, of pleural effusion. Often, after careful medical evaluation including repeated examinations of the pleural fluid, the etiology remains indeterminate. In some reported series, this indeterminate group runs as high as 38 per cent. Furthermore, in both groups of cases, those of established and those of unestablished etiology, the control of pleural effusion can be difficult.

The purpose of this paper is to describe the rationale of open thoracotomy with elective parietal pleurectomy in the management of certain cases of pleural effusion. Open thoracotomy usually provides a direct approach to the diagnosis. Parietal pleurectomy with resultant obliteration of the pleural space may then be justifiably carried out providing the attending circumstances suggest a reasonably long and comfortable patient survival.

This procedure has been used in seven patients, in six of whom the cause of the effusion was indeterminate before surgery. Three of the effusions proved to be caused by metastatic malignancies and three by inflammatory diseases including tuberculosis. All seven of these patients have been closely followed postoperatively. In none has there been a re-accumulation of fluid. In two cases survival time was too short to have justified the procedure.

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CARCINOMA OF ABERRANT BREAST OF THE AXILLA

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Having had a patient with proven carcinoma of an aberrant mammary gland in the axilla, we were surprised at the paucity of literature available for a study of the subject. It seemed worth while to review the more recent literature and to relate our experience with this condition.

Aberrant breasts come under the general classification of polymastia. This latter condition in the human is characterized by the presence of more than two breasts (Dorland).[8] Under this general heading a differentiation is made between supernumerary breasts and aberrant breasts. Supernumerary breasts have a nipple, areola, or both, with or without atrophic breast tissue, occurring along the nipple line, usually below the breast and can extend down as far as the vulva. Aberrant breasts consist of breast tissue having no nipple or areola and are usually found in the axilla but may occur less frequently in the infraclavicular or juxtasternal regions and have been reported in the vulva.[12]

The incidence of aberrant breasts is not known. Examination of two thousand women observed in a Tumor Center established the incidence at approximately 6 per cent.[3] It is well established that the incidence of carcinoma occurring in supernumerary breasts is low. There is evidence, however, that axillary breast tissue is more prone to malignant changes than normal breast tissue.[1, 17] It was observed by Matti[14] that aberrant breast tissue showed a considerably higher tendency toward malignancy than supernumerary mammary glands which are no more prone to malignant change than normal glands. Carcinoma was found in 3.8 per cent in a series of 26 operated cases of aberrant breast of the axilla by deCholnoky.[3] The most common tumors found in aberrant breast tissue are cancer and fibroadenoma, with the former being the more frequent.[5]

Aberrant breasts occurring in the axilla may remain asymptomatic but frequently give rise to complaints which first become apparent at the time of the beginning of the menses when the patient notices an axillary fullness, at times mildly painful, and occurring in association with the menstrual cycle. At this time the symptoms are mild. At times the individual first becomes aware of a mass in the axilla about the fourth month of pregnancy, or in some cases not until the beginning of lactation at the termination of pregnancy. We have observed one patient who had not had a pregnancy who first noted an axillary tumor and tenderness associated with fibrocystic change in aberrant breast tissue which was also present in the mammary glands.

There is no general agreement as to the potential danger from cancer in these aberrant tissues. Dexter and Dexter[6] write, "The instance of neoplasm in aberrant breast tissue in the axilla is apparently less than in the normally placed breast." In a review of the literature, Storey[21] noted that many American and English authors were of the opinion that carcinomatous changes in accessory breasts occurred rarely. Viewing this with alarm, he calls attention to the study made by Rebeiro,[18] who found numerous references to the tendency of these tumors to undergo malignant change. Storey did not differentiate between aberrant mammary glands and supernumerary mammary glands in his review, and this factor may explain why American and foreign authorities are not in accord. The true incidence of aberrant breasts is probably not known but has been estimated to occur in 1.7 per cent in humans and is more common on the left side.[15]

The few series of patients reported with aberrant breasts with little or no instance of carcinoma were on the average in an age group much lower than the period when cancer usually manifests itself in breast tissue. In Geschichter's[11] series, there were seven instances of carcinoma in aberrant breast tissue, all occurring in the axilla. The ages of the patients varied from 39 to 69 years of age. Cole[4] believes that the occurrence of malignant changes in these tissues is not unusual. In collecting all cases of aberrant mammary tumors reported up to 1929, Razemon

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and Bizard[17] found 43 malignant tumors in a total of 76 cases. One was in a male patient. An adequate follow-up was possible in only 19 cases. In 13 cases operated upon with preservation of the breast and an axillary dissection, there was recurrence in 10 cases 6 weeks to 2 years after operation. The other three of this series were not observed long enough to evaluate the outcome. Four patients had a radical mastectomy in conjunction with the excision of the aberrant breast, and the results of these cases were no better. Three of these patients showed marked dissemination or were dead of the disease in less than 2 years, and the remaining case had only been followed 5 months. In the other two cases, the surgical procedure was not known, but metastasis had appeared in both instances. Erdmann[9] reported having operated upon seven aberrant breasts, three of which were highly malignant, one being in a 26-year-old man. In all of these, metastases later occurred.

"Oversight in recognition of the true nature of these tumors may be due to failure to observe them because of the position of the patient on the examining table. When the patient is lying supine it is possible that it may be overlooked as aberrant tumors retract under the pectoralis muscles. The swelling is obvious when the patient is upright with the arms abducted."[19]

Irritation resulting from stagnation of secretion and cell debris has been believed to be one of the etiologic factors in breast cancers of mice.[7] This would pertain to aberrant breasts which have no excretory ducts and would account for the greater frequency of cancer in aberrant breasts. Roberts has asserted, "The susceptibility of this tissue to malignant tumor formation, its cyclic swelling and painful character, are indications for surgical excision. All nodules bordering the breast and axilla under the clavicle or over the sternum should be held in mistrust and excised."[19]

A 40-year-old white woman entered the hospital on July 8, 1956, for the excision of axillary masses. A week before entering the hospital the patient's family physician had removed a purplish discolored mass from the right axilla, which apparently was attached to the skin and underlying structures, which was diagnosed as carcinoma of breast tissue. The patient had an active duodenal ulcer and during the past 6 months had been seen by two physicians who had assured her that the mass in the right axilla was innocuous and needed no medical or surgical care. The patient stated that she had noticed the bilateral axillary masses since the beginning of puberty and had noticed discomfort secondary to swelling and tenderness during her menses which had become more pronounced during the past several years.

The patient was operated upon on July 9, 1956, at which time the left aberrant breast was excised. The right aberrant breast was excised, including the skin surrounding the area of the previous excision with all the lymph nodes and fatty tissue from the axilla as well as the tail of the right breast. The remainder of the breast was left intact. The pathologist's diagnosis read as follows: "Adenocarcinoma of aberrant (right axillary) mammary gland with metastasis to regional lymph nodes; aberrant mammary gland, each axillary region." The patient recovered and was given x-ray therapy in the region of the right axilla in August 1956.

In December 1957 the patient was hospitalized for pathologic fractures of several ribs, and a bilateral oophorectomy was performed with rapid improvement of pain. She was treated with testosterone 50 mg. to 100 gm. three times weekly, and N,N,N-triethylenethiophosphoramide (Thio-TEPA). The patient was comfortable, doing light housework on a ranch, until August 1958, when she was rehospitalized for recurrence of chest pain. She died on September 19, 1958. Autopsy showed extensive metastatic lesions of the skeletal systems, pleura and suprarenal bodies, but no evidence of infiltration or metastasis to the breast on the side of the original tumor and no evidence of recurrence in the original tumor site.

Ravdin[16] reported on aberrant breast carcinoma of the subclavicular region with later metastasis. Radical mastectomy was done but examination showed no breast involvement. Schmidt-Tannwald[20] has reported a similar case, and others have been recorded.[13] Falk[10] described an aberrant breast carcinoma in the sternal region, the tumor being excised locally and the tumor area being treated with x-ray therapy. The patient later manifested recurrence and generalized metastasis.

A white woman, aged 69, entered the hospital in July 1948, for the treatment of a tumor of the midsternal area which she believed had been present for a number of years but had recently

been growing. On entrance to the hospital it was found to be about 2 cm. in diameter. Excision of the tumor was done under general anesthesia, and by microscopic examination a diagnosis of carcinoma of aberrant breast was made. No masses were palpated in either breast or in either axilla. Postoperatively the patient was given x-ray therapy to the sternal area. The patient was readmitted to the hospital in 1950 at which time a laparotomy showed papillary adenocarcinoma of both ovaries with metastasis to the peritoneum. She developed progressive ascites and died in February 1952. Examination at the time of death showed no evidence of recurrence of the aberrant breast carcinoma of the sternum or spread to either breast or axilla, her death having been caused by the carcinoma of the ovaries.

It seems obvious from the discussions in the literature that carcinoma of aberrant breast is a virulent disease and its onset is insidious. From our studies and limited experience it would appear, as does usual breast cancer, to be more morbid in younger than in older individuals. It is also noted that the metastases apparently occur early and spread by the lymphatics and the blood stream. In view of these facts we would agree with those authors who advocate a prophylactic excision of aberrant breasts.

We believe that excision of the axillary tumor including all the lymph nodes and tail of the breast is all that is surgically indicated when aberrant breast cancer in the axilla exists. In those reported cases, where an associated radical mastectomy was done for aberrant breast carcinoma of the axilla, the results were no better than those with the more conservative excision of only the aberrant breast cancer with the axillary contents and tail of the breast. A wide excision of the sternal aberrant breast cancer associated with local x-ray treatment seemed to be the best therapy as these tumors most likely have metastatic migration to the mammary chain lymph nodes rather than to the breast or axillary nodes. In the two cases we have described there was no evidence of spread from the aberrant breast carcinoma to the adjacent breast tissue.

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Editorial

BASIC TRAINING IN SURGERY

In 1952, we discussed in this column the concept that surgery in America should be performed by surgeons, meaning thereby, by physicians who have taken sufficient additional graduate training to become experienced and skilled in the operational treatment of disease or injury. Today there is much discussion of this basic concept as it relates to the recognized surgical specialties and resolves itself around a growing interest in the question: should not the surgical specialist be a surgeon first, and a specialist thereafter? Specifically, how much training in general surgery should the surgical specialist have before focusing his attention on a single organ system, so that in his case too, surgery would be done by a broadly trained surgeon?

There need be no single answer to this question. Indeed, the answer may properly be different for each specialty, insofar as it reflects the actual nature of practice in that specialty. Thus, for example, the practice of ophthalmology is largely nonoperative, and even for the 10 per cent or so of patients requiring surgery, the procedures are very specialized and rarely involve more than the orbit and its contents. The patients are rarely very ill, and the surgery is essentially nontraumatic. Urology, on the other hand, might be considered to stand at the opposite pole of experience. Modern urology is largely operative and is involved with problems and procedures of increasing complexity, problems which often involve the general care of the very sick patient and which may require surgical techniques to deal with organ systems other than the genitourinary tract, as for example, intestinal reconstruction of ureter or bladder, or vascular anastomosis for renal transplantation. Extracorporeal dialysis in the treatment of renal failure requires a much broader and deeper understanding of body physiology than was necessary in the past, when a major portion of urology was the manipulative local treatment of urethral stricture! Surely, then, the needs for basic training in general surgery for the modern ophthalmologist and the modern urologist must be quite different. The other specialties may fit in intermediate positions on the scale.

There appears to be little agreement even within specialties concerning the requirements for training. The need for some progress toward more uniformity of opinion is made especially evident when one notes the very large number of training programs involved. The resident programs in hospitals approved by the Conference Committee in 1960 and 1961 are shown in table 1.

As this extensive number of training programs has developed, largely in the last 15 years since World War II, two aspects of the programs are noteworthy and pertinent. These are: (1) a trend toward prolonging the training period, particularly in the specialties; and (2) the growing recognition that lack of basic surgical training is sharply limiting the area of competence of many of the current practitioners of the specialties. This lack has even, on occasion, limited progress in a specialty. For example, the urologist's lack of familiarity with standard vascular techniques is perhaps one reason why progress in the problem of transplantation of the kidney has been achieved by general and plastic surgeons, rather than by urologists; and currently the treatment of renal artery occlusive disease as a cause of hypertension is being evaluated, again by general surgeons rather than by the specialist. It appears that there is still room for improvement in our specialty resident training programs.

The lack of uniformity in requirements for general surgical training in preparation for the surgical specialties is evident from the data of table 2.

In a report at the San Francisco meeting of the American College of Surgeons, Dr. Frank Glenn suggested a possible approach to the solution of these problems. It was proposed first, that 2 years of general surgical residency training be a prerequisite for acceptance into an approved specialty training program and second, that 2 additional years of training in the specialty would complete the minimal requirements of that Board.

Dr. William Scott reported to the Graduate Training Committee of the American College of Surgeons that although widespread discussion of this problem by many high level committees and

TABLE 1

Resident programs in hospitals approved by Conference Committee

	Programs	Appointments Offered
General surgery	704	5,837
Neurosurgery	115	354
Obstetrics-gynecology	477	2,600
Ophthalmology	165	763
Orthopedics	286	1,187
Otolaryngology	131	510
Plastic surgery	52	119
Proctology	13	29
Thoracic surgery	97	236
Urology	227	729
Total	2,267	12,364

TABLE 2

Training requirements by various surgical specialty boards

	General Surgery Prerequisite (or Recommended)	Training in Specialty (years)	Total Post-intern Training (years)
Anesthesiology	None	2	2
Neurosurgery	1–2	4	5
Obstetrics-gynecology	None	3	3
Ophthalmology	None	3	3
Orthopedics	1	3	4
Otolaryngology	1	3	4
Plastic surgery	3	3	6
Proctology	3	2	5
Thoracic surgery	3–4*	2	5–6
Urology	1	3	4

* Certified by ABS.

surgical boards indicated wide differences of opinion, there was some unanimity in the recognition of the mutual interdependence and interlocking needs of general surgery and the surgical specialties in residency training. There was great divergence of opinion as to how this interrelationship could best be achieved. Dr. Scott's subcommittee recommended, nonetheless, that a 2-year basic residency in general surgery should precede and be a prerequisite to completion of training in both general surgery and the specialties. Just such a program has recently been instituted at the University of Colorado Medical Center.

Further opinions on this proposal will probably be sought by the American College on a broad scale from the directors of general surgery and specialty training programs. In answering such a questionnaire, it is to be hoped that these directors will recognize the mutually interlocking needs of general surgery and the specialties in residency training, and will remember that progress in medicine today is made on a broad, usually multidisciplined base, and that above all, for the good of the patient, surgery should be performed by surgeons, including the surgical specialist. Opportunity for such training, therefore, must be made available to him by the directors of general surgical training programs, and the specialty boards must be willing to decrease the required time of training in the specialty for those who have acquired basic training in surgery.

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Book Reviews

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Outline of Orthopaedics. By JOHN CRAWFORD ADAMS. The Williams & Wilkins Company, Baltimore, 1960.

The author's goal in writing an *Outline of Orthopaedics* was to provide a source of information for the student and general practitioner. The material presented covers the scope of orthopedic problems. The second chapter is of particular value in classifying orthopedic disorders. The bulk of the text is devoted to a discussion of the musculo-skeletal disorders, by region. Of particular value are the illustrations on physical examination and methods of demonstrating abnormalities. The reproduction of x-rays is good and the individual films are well chosen to illustrate a point.

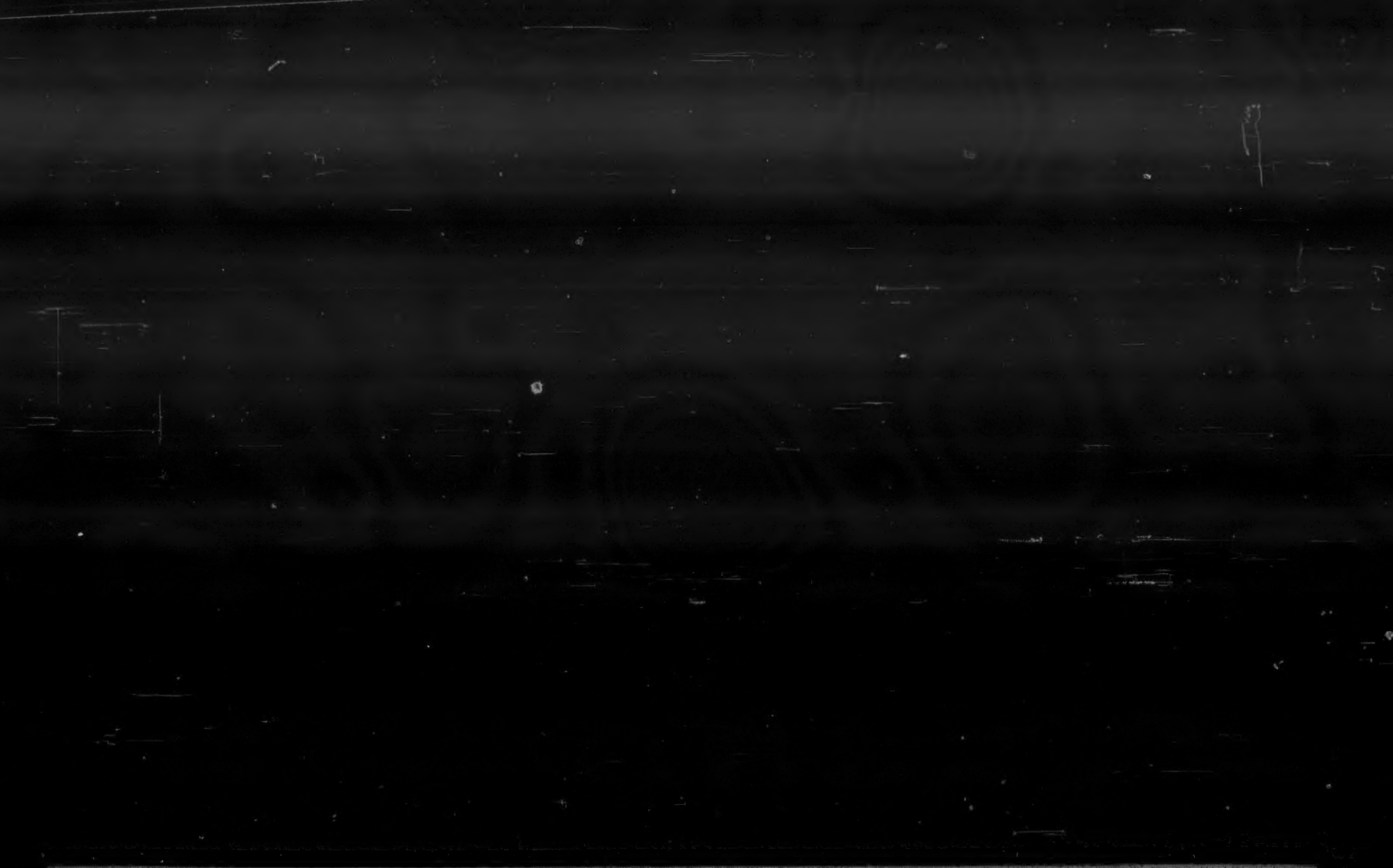
It is felt that this book is of particular value to those unfamiliar with orthopedic disorders, for it provides a good starting point for the understanding of this specialty.

A. GIBSON PACKARD, JR., M.D.

The American Academy of Orthopedic Surgeons, Volume XVI, Edited by FRED C. REYNOLDS, M.D. C. V. Mosby Company, St. Louis, Mo., 1959.

The 1959 Instructional Course Lectures is a completely clinical edition of orthopedic surgery. In the past there have been articles of a research or academic nature and most of these articles represented significant contributions to the field of orthopedic surgery.

This volume should be of interest to all practitioners of surgery, especially those who feel to any extent the need of enlightenment in trauma. Although no single volume of the Instructional Course Series constitutes an entire orthopedic text, the series, since their inception, certainly represents the widest and most comprehensive coverage in the field of orthopedic surgery. For instance, this particular volume has no specific section on bone tumors, whereas in past years, a good portion of the book may have dealt with the pathologic aspects of bone and soft tissue tumors. Needless to say the individual articles are extremely well written, usually by recognized authorities in their particular fields. They are well illustrated and diagrammed.

This particular volume would be of use to any general surgeon, and for those of us who practice orthopedics, or for those residents who some day will be in practice, this volume and this series of yearly volumes is a *must*.

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